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THE MANAGEMENT OF EARLY BREAST CANCER.

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THIS paper attempts to assess without prejudice the relative places of surgery and radiotherapy in the treatment of potentially curable breast cancer. Although radical mastectomy has become firmly established as the treatment of choice for these patients, during the past decade there has been a greater recognition of the part that can be played by radiotherapy. Moreover, there has arisen an increasing awareness of the unsatisfactory results of surgery in large numbers of cases, with the result that many people now feel puzzled and undecided. On the one hand, one hears the viewpoint expressed (not infrequently by pathologists) that it hardly matters what type of operation is done, because if widespread dissemination is to occur it will have occurred early. On the other hand, one meets the teaching that, if only the operation is sufficiently radical, embracing not only the internal mammary nodes but also the mediastinum and supraclavicular regions, the patient is likely to be cured. It is obvious that the truth lies somewhere between these two extremes.

Selection of Patients for Radical Mastectomy.

Radical surgery, providing it is reasonably conceived, and does not carry in itself a prohibitive mortality, remains the treatment of choice for the majority of patients. It is quite clear that the statistical results of such surgery will depend on which patients have been selected as suitable for operation, and which rejected. Indeed, there exists a wide variation in opinion concerning what constitutes operability, as the following examples show. Of a series of 100 patients referred to the Peter MacCallum Clinic for post-operative radiotherapy, axillary metastases had been found present in 64; but the actual number with node involvement was almost certainly higher, since in many of these cases sections were prepared of not more than two nodes. Riddell (1948), at St. George's Hospital, London, reported axillary metastases in 79% of one group of patients subjected to operation, whereas Haagensen, at the Presbyterian Hospital, New York, found such metastases in only 58% in his personal series (Haagensen, 1956). The significance of this latter figure is emphasized by the fact that it was obtained after an examination of an average of 37 nodes per patient. This may explain why Haagensen can produce excellent statistical results for radical mastectomy—even a 65%

five-year clinical cure rate in early Stage II patients with one or two nodes involved, and a 40% five-year survival rate for all Stage II patients. Andreassen *et alii* (1954) have carried selection even further, and have reported the completion of a series in which axillary nodes were involved in only 41%.

For the correct evaluation of this problem there must be a recognition, not only of what can be achieved by modern radiation therapy, but also of the poor results which follow indiscriminately applied surgery. If radical mastectomy is to maintain its place, the criteria for its performance should be stricter and more rigidly applied than they have been in the past. Operations should not be performed on patients in whom the disease has already spread beyond the limits of tissues which can be removed. When carcinoma has invaded the nodes at the apex of the axilla, the supraclavicular nodes or the nodes of the internal mammary chain as high as the first intercostal space, no form of surgical attack is likely to save the patient.

Estimation of Degree of Spread.

Some errors of judgement must inevitably continue to occur. A small apparently localized carcinoma may have already become widely disseminated by the blood-stream. Again, there may be mediastinal invasion via the internal mammary chain of a degree which is irremovable by any operation, even one of the extended type.

Handley's most recent report on internal mammary node biopsy in 250 cases of operable breast cancer shows that with inner quadrant tumours, metastatic deposits were present in the internal mammary nodes in 45% of cases (Handley, 1958). Similarly, Urban, in a series of 300 patients with operable carcinoma of the breast, found 58% of those with affected axillary nodes also to have internal mammary node involvement (Urban, 1959). Moreover, there may be unsuspected metastases in the supraclavicular nodes. Andreassen *et alii* (1954), in a series of 98 cases in which surgery was extended to this region, found unsuspected metastases in 33% of cases in which the axillary nodes were invaded.

Thus, we must recognize that the disease is almost always more advanced than it appears, and an accurate estimate of its extent is usually impossible by clinical means alone, especially in obese patients, in whom the most careful examination of both breast and axilla may be hopelessly in error. Many patients are seen in whom there is considerable doubt as to whether the axilla can be cleanly dissected, or whether upward extension has rendered this impossible.

Apex of the axilla exposure and inspection, a modification of the apical biopsy advocated by Haagensen, is a simple procedure which can precede a proposed radical mastectomy. One of us (T.H.A.) has made use of this and now proposes to apply it to all such doubtful cases (Figure I).

A three-inch incision, which can be made part of the incision for the proposed radical operation, is made below the middle of the clavicle. The pectoralis major is split at the junction of the sternal and clavicular fibres, so that the fascia above the pectoralis minor is exposed (Figure II). The axillary vein medial to the thoraco-acromial vessels is then found. If obviously enlarged nodes are seen to surround and adhere to the axillary vein at the level of the first rib, the patient should be spared the mutilation of an operation which will be useless in arresting the disease.

It is not suggested that the finding of a single small invaded node just above the level of the pectoralis minor should necessarily forbid radical mastectomy; opinions will vary as to what constitutes inoperability. However, all will admit that hopelessly incurable invasion of this region can easily be recognized. Such a condition is only too often an unexpected operative finding at a stage in the operation when there can be no turning back. For those who care to add internal mammary biopsy to this, the finding of involved nodes at this site must contraindicate an operation other than one of the extended type.

If such exposures or biopsies are not performed, radical mastectomy will not prove a curative operation in about one-third of clinical Stage I cases, and in about two-thirds of clinical Stage II cases.

Criteria of Operability.

In spite of these implications that purely clinical methods may be inadequate, there should always be the most painstaking and meticulous examination of the breast, chest wall and gland fields for signs which may indicate that radical mastectomy is contraindicated for purely local reasons. No apology need be made for stating the following alarmingly long list of findings, any of which is sufficient to declare a patient inoperable.

Skin:

(*a*) Wide attachment or wide ulceration.

(*b*) Wide *peau d'orange* or extensive œdema of skin over the breast.

(*c*) Satellite nodules in the skin over the breast or the presence of parasternal nodules.

Breast:

(*a*) All four quadrants involved.

(*b*) Carcinoma of "inflammatory" type.

(*c*) Deep fixation to pectoralis muscle or chest wall.

(*d*) A lump in the opposite breast which is clinically malignant.

Nodes:

(*a*) Fixation of axillary nodes.

(*b*) Papable supraclavicular nodes.

(*c*) Enlarged nodes in the opposite axilla.

Arm:

Œdema of the arm.

Probably the commonest errors are the overlooking of very small supraclavicular nodes situated immediately above and almost behind the clavicle, and the acceptance of a wide area of *peau d'orange* and breast œdema as not contraindicating operation.

Policy in Early Cases.

Stage I Cases.

In Stage I cases (in which the breast tumour is relatively small with minimal or no skin involvement, and no axillary nodes are palpable), radical mastectomy is advised. The following facts must be borne in mind if biopsy of the apical axillary or internal mammary nodes has not been carried out as a preliminary:

1. In 15% (Berven, 1949) to 50% (Riddell, 1948), the axillary nodes may be found involved, although they were not palpable before operation. The striking differences between these figures must be due partly to differences in clinical judgement, and partly to the fact that there is a good deal of variation among pathologists in searching the operation specimen for foci of carcinoma in the nodes. There is very little doubt that the usual methods employed will miss lymph-node metastases in a considerable proportion of cases. Few pathologists would have the time and assistance in their laboratories to enable them to equal the thoroughess of Pickern, of the Presbyterian Hospital, New York, who, using a clearing technique for finding nodes, examines an average of 37 per case (Haagensen, 1956).

2. In 15% of cases in which the axilla is clear, the internal mammary nodes are likely to be involved (Urban, 1959).

3. In a small percentage, the supraclavicular nodes may be invaded although the excised axillary nodes are reported clear.

4. According to Smith (1958), the examination of washings from under the mastectomy flaps at operation gave positive results for malignant cells in 26% of 120 cases and aroused suspicion in a further 14%. Smith noted that these figures could be later correlated with the development of metastases in these two groups.

Thus, in about one-third of apparently Stage I cases there may be metastases in the local drainage areas, and

in a further minority there may be free malignant cells under the scar. For these reasons, it is recommended that post-operative X-ray therapy be given as soon after surgery as possible.

Stage II Cases.

In Stage II cases (in which the primary tumour is as in Stage I, but the homolateral axillary nodes are palpable and mobile), radical mastectomy is again advised. Nevertheless, the following facts are relevant:

1. In this stage, 58% of patients are likely to have internal mammary node involvement —61% in the case of medial lesions and 51% in lateral lesions (Urban, 1959).

2. In 33% of these cases, supraclavicular nodes are likely to be involved (Andreassen *et alii*, 1954).

Thus about two-thirds of these patients are likely to have metastases in the surgically untreated drainage

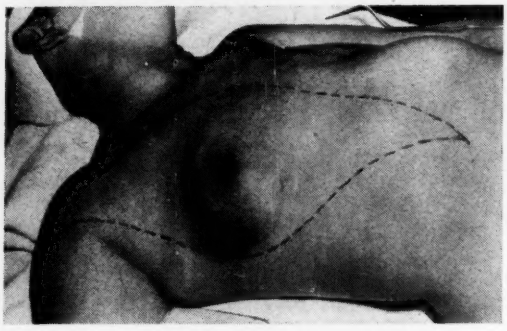

FIGURE I.
Incision for apical biopsy as part of incision for radical mastectomy.

areas, and the likelihood that malignant cells may be disseminated under the scar is as in Stage I. Again, post-operative X-ray therapy is recommended.

Place of Extended Operation.

Undue enthusiasm and vehemence concerning extended operations are at the present time undesirable, although large numbers of these operations have been carried out in different centres. The extreme procedure carried out by Wangensteen and Lewis at the University of Minnesota includes a supraclavicular dissection and internal mammary and mediastinal dissection, as well as a conventional radical mastectomy. It carries an operative mortality rate of 13%, which renders it completely unreasonable and unjustifiable. On the other hand, the operation performed by Jerome Urban at the Cancer Memorial Hospital, New York, is more realistic and can be performed without adding appreciably to the operative mortality and morbidity. A preliminary step is inspection of the first intercostal space, and if it is seen that malignant nodes are present and one cannot ligate the internal mammary vessels above them, the operation is contraindicated. Otherwise the pleural cavity is opened, and parts of the second, third, fourth and fifth costal cartilages are removed with the internal mammary vessels and nodes undisturbed on their deep aspects. This tissue is removed in continuity with the main radical mastectomy specimen. One of us has had some experience of this procedure, reserving it strictly for patients with early carcinoma situated in the medial and central portions of the breast when the patient is reasonably young and in good general condition. It seems undesirable to apply it to lesions of the lateral half of the breast, since they spread to the internal mammary nodes less often, and later in the course of the disease. Urban's latest report, reviewing 300 patients treated by this procedure, shows that 33% of the patients with invaded internal mammary nodes are clinically free of disease more than five years after operation (Urban, 1959). These would appear to be important figures which call for serious consideration of this operation in certain cases of breast carcinoma.

Simple Mastectomy.

A good deal of prominence has been given to the teaching of McWhirter and others that simple mastectomy followed by irradiation gives results as good as those of radical mastectomy. The axilla is not dissected, either because it is thought that an attempt to do so will be futile, or in the belief that dissection will disseminate the tumour cells.

It is important to recognize that patients so treated by "simple" surgery usually receive both X-ray therapy and ovarian ablation therapy in addition, with the result that useful palliation is achieved. Moreover, the final

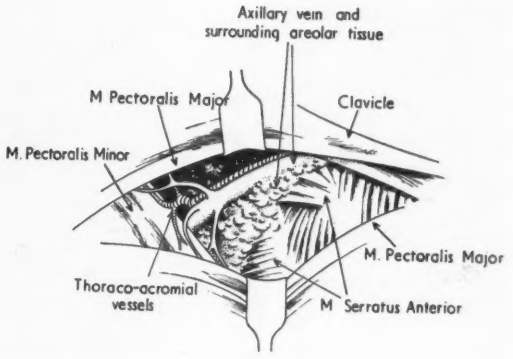

FIGURE II.
Exposure of axillary apex.

results, when expressed in figures, may well seem not inferior to the results of radical mastectomy when the operation has been performed indifferently and without proper selection of cases. However, it seems remarkable that the poor results of indiscriminately performed radical mastectomies should lead some surgeons to abandon the radical operation altogether, instead of choosing their patients more critically. McWhirter, Williams and Murley have condemned an operative procedure because at certain hospitals good results had not been obtained from its use.

The patient has nothing to gain, since in both cases she loses her breast. The morbidity of both operations is practically the same, and the mortality for radical mastectomy when performed under good conditions of surgery and anæsthesia should be nil. On the other hand, the patient has much to lose. If she has only a limited axillary involvement, the choice of simple mastectomy may determine her death, since radiation cannot destroy axillary metastases with any certainty.

Simple mastectomy is, however, a useful palliative operation for the type of ulcerating carcinoma which often grows slowly in elderly patients (Figure III). Not infrequently, these neglected tumours are found to be quite mobile, and both the patient and relatives are grateful for the removal of an offensive lesion (Figure IV). A quick and uneventful convalescence follows in most cases, and it is found that the operation can be quite easily conducted with the minimum of disturbance to a patient who, being in the seventh or eighth decade, would find 20 daily visits for irradiation a greater physical burden.

The Scope of Post-Operative X-Ray Therapy.

Mammary carcinoma is in the main only a moderately radio-sensitive tumour. This means that the cancericidal dose of X rays is of a similar level to the maximum dose which can be tolerated by the normal tissue. This

cancericidal dose for mammary cancer has been established by observing the microscopic response of primary breast tumours or of metastatic skin nodules to various doses of irradiation.

Now it is a fundamental principle of radiation therapy that the larger the volume of tissue irradiated, the smaller the dose that can be tolerated. The reason is that the tissue tolerance essentially depends on the effect upon the blood vessels in the irradiated area. As a result, if a large volume is irradiated, the dose of irradiation necessary to sterilize the tumour in the majority of cases of breast cancer will lead to a severe skin reaction and possible necrosis. For this reason, only a subcancericidal dose can be achieved in the majority of patients given pre-operative irradiation, and in the case of widespread irradiation of the chest wall, axilla and supraclavicular and internal mammary areas after operation.

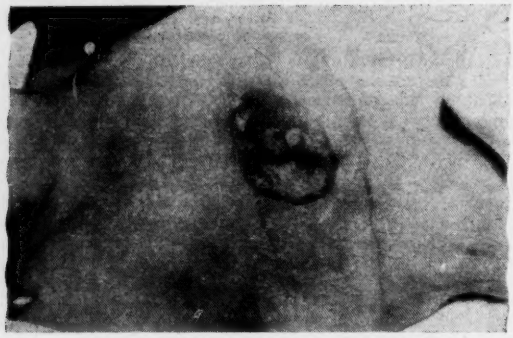

FIGURE III.

Scirrhous ulcerating type of mammary carcinoma suitable for palliative simple mastectomy.

If, however, the volume irradiated is smaller, the body tissues will tolerate a higher dose, and by this means a cancericidal level may be achieved.

Irradiation after radical mastectomy is therefore, in our opinion, best confined to the surgically untreated but vulnerable lymphatic drainage areas—that is, the apex of the axilla, the supraclavicular draining areas and the internal mammary draining areas. This reduces the area of irradiation from approximately 600 to only 200 cm.[2] and allows a correspondingly higher dose to be achieved.

It must be understood, however, that the dose of radiation which can be achieved with wider field irradiation has been proved cancericidal in a minority of cases. Such a dose will, at the very least depress mitotic activity if given pre-operatively, and after operation will stimulate fibrosis in the scar so that viable malignant cells may be incarcerated in fibrous tissue.

Lymphatic Drainage Areas.

The location of these defined nodes does not permit them to be adequately treated by a continuous field with the use of conventional X-ray therapy. For this reason, the internal mammary chain is treated as one unit, and the supraclavicular and axillary areas are treated as a contiguous unit. Both the supraclavicular and the internal mammary chains of lymphatics terminate in the jugulosubclavian venous confluence of lymphatic trunks or terminate in the sentinal supraclavicular node lying behind the head of the clavicle. The latter is included in the supraclavicular-axillary field. (See Figure V.[1])

Certain groups of nodes can safely be ignored in a post-operative "prophylactic" irradiation. Adequate radical mastectomy removes lateral pectoral nodes cleanly and dissects out the contents of the axilla. Recurrence presenting in the dissected axillary or subscapular area is extremely rare, whereas supraclavicular recurrence is common. Metastases in the lower deep cervical nodes suggest retrograde lymphatic embolus and therefore the likelihood of disseminated disease. Irradiation of this area could not be regarded as part of a planned treatment for a potentially curable patient.

The supraclavicular-axillary area requiring irradiation is therefore considered to be a continuous chain of nodes extending from the sterno-clavicular joint to the mid-axilla, following the line of the axillary vein. The area is established by a venogram and irradiated with a margin of 2 to 3 cm. above and below the line of the vein (Figure V). It is essential to be accurate because of the narrow volume irradiated. The inaccuracy of clinical estimates of the position of the nodes has been established by us, with venograms checking estimates based on surface anatomy.

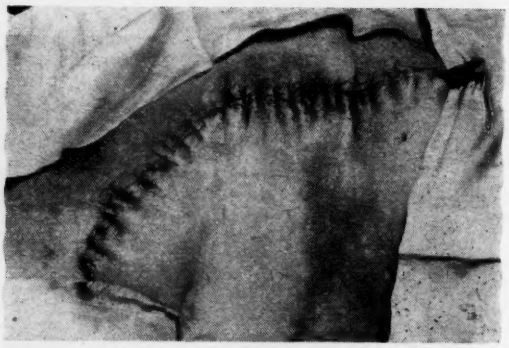

FIGURE IV.

Same case as in Figure III, after simple mastectomy.

The internal mammary field extends down to and includes the sixth intercostal space. Two oblique fields of irradiation are used (Figure VI) centred on a line 2 cm. from the mid-line, and a width of 4·5 cm. is effectively irradiated at a depth of 1·5 to 2 cm. The dose falls off very rapidly at a depth with this technique in order to avoid irradiation of mediastinal structures. The skin reaction from this technique is illustrated in Figure VII.

Chest Wall.

The skin flaps are not treated as a routine. Most authorities report recurrence in the skin flaps in approximately 10% of cases, in spite of the much higher proportion of positive findings in mastectomy washings noted above. Thus, post-operative irradiation of the chest wall as a routine may possibly benefit one patient in ten, but limits the possible dosage to the more vulnerable sites—that is, the lymphatic drainage areas.

With regard to the 10% of patients who do develop later recurrence in the chest wall, it is important to differentiate recurrences as of four types according to their site of origin, as follows: (*a*) arising in the parasternal region from the internal mammary nodes; (*b*) arising in or near the suture line; (*c*) arising at a distance from the suture line, but within the skin flap or graft area; (*d*) arising outside the skin flap area.

Recurrence arising in the internal mammary nodes suggests the likelihood of their involvement before operation, and in any case this area is always irradiated after operation as a routine. Recurrence in the suture line is generally due to implantation, and is often solitary. If this is so, surgical excision or localized irradiation is usually adequate for cure.

Both (*c*) and (*d*), which together represent less than 5% of cases, are a sign of a lymphatic permeation and usually herald widespread recurrence. It is not likely that these patients can be treated curatively in any case. Thus, after adequate radical mastectomy, careful and

[1] For Figure V, see art-paper supplement.

frequent follow-up investigation to detect the first evidence of recurrence in the scar, and then its treatment should cause no worse a prognosis than that which follows routine post-operative irradiation of the scar. However, it is the practice of the Clinic to carry out post-operative "prophylactic" X-ray therapy to a limited area of the skin flaps in some cases in which there is a suspicion that an insufficient margin of tissue has been excised. The following groups provide examples: (i) when the primary tumour was larger than 5 cm. in diameter; (ii) in the case of inframammary or medial quadrant tumours; (iii) when the pre-operative description indicates wide invasion of the skin or early deep fixation.

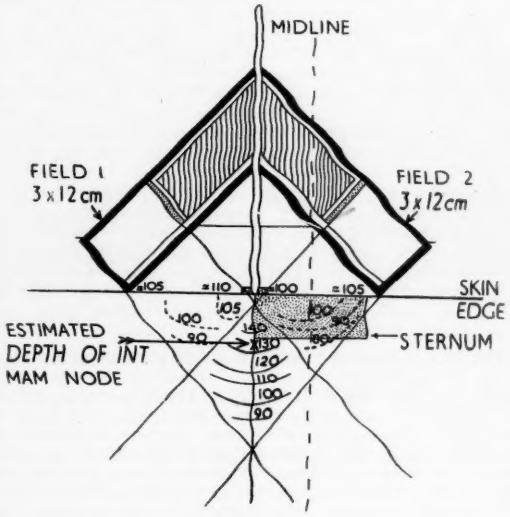

FIGURE VI.

Distribution of dosage with the use of oblique fields.

In these groups, the likelihood of local recurrence is high after injudicious surgery. However, when patients have been strictly selected for surgery and wide, adequate margins of tissue removed, the skin flaps are not irradiated.

Pre-Operative Irradiation.

Pre-operative X-ray therapy is strictly limited to "operable" patients, and it must be emphasized that an inoperable tumour cannot be made operable by a course of X-ray therapy. We have used the technique in a number of late Stage II cases, but consider that the apex of the axilla should, in this type of case, be examined by biopsy as a preliminary step, before a decision as to subsequent surgery is made.

We know that the dose that can be achieved by pre-operative X-ray therapy is in the majority of cases not cancericidal. It is true that we have had a small proportion of cases in which, after pre-operative irradiation, no demonstrable tumour tissue was detected at subsequent operation; but in the majority we can achieve only depression of mitotic activity.

The growth restraint that is visible macroscopically after irradiation in these cases is reflected microscopically by severe chromosomal damage in about half the cases, and by moderate or slight changes in the others. It is a generalization that the more anaplastic the tumour, the more sensitive it is to irradiation; but the dose of irradiation given, must be subcancericidal to the majority of tumours in order to avoid causing surgical difficulties later. Nevertheless, the fact that the tissues have an intact blood supply at irradiation should cause a greater radiobiological effect than would the same dose upon devitalized tissues after operation.

Regrowth of the tumour is usually visible microscopically four to six weeks after the end of treatment, and it is therefore recommended that mastectomy be carried out within four weeks of completion of the treatment—that is, as soon as the skin reaction settles. The X-ray dose level to the skin is deliberately limited, and our experience is that limiting the skin dose to a maximum of 3800 rads (compared with 4000 rads in four weeks in post-operative therapy) is followed by normal healing of skin flaps, and by no difficulty from increased bleeding at operation. As was stated before, this is the maximum dose tolerated, but is not optimal as far as its cancericidal effect is concerned.

Pre-operative irradiation is, therefore, indicated in late Stage II cases in the presence of multiple large axillary nodes, when selection by a preliminary biopsy from the apex of the axilla has demonstrated that upward invasion has not yet reached this high level.

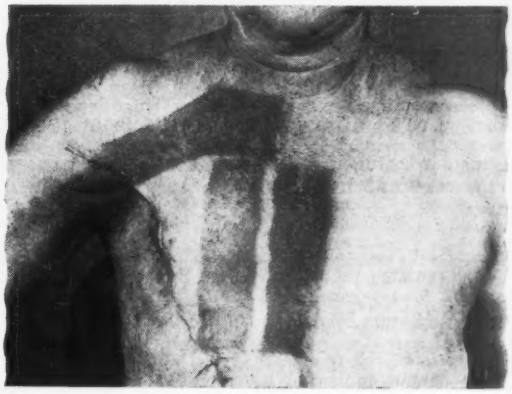

FIGURE VII.

Typical skin reaction from technique shown in Figure V.

After irradiation, the primary tumour is usually found to diminish in size rapidly, so that when the operation is performed, the surgeon must have in mind the original, rather than the present, limits of the tumour. Skin tattooing carried out before irradiation is recommended as a useful aid in this regard. It is evident that in patients who receive pre-operative irradiation, the skin will not tolerate post-operative therapy.

Radium implant or radical X-ray therapy is sometimes used in the treatment of early breast cancer in "poor risk" cases or when operation is refused. Either may also be useful in senile patients with slowly growing lesions.

The Role of Castration.

It is well established that surgical castration may induce a long remission in pre-menopausal patients, even in the presence of advanced disease. Various authors quote objective relief and remissions in 40% to 50% of cases. Radiation menopause has been used for the same purpose, and here again various series report 30% to 55% of remissions resulting from this procedure. Nevertheless, it is generally agreed that a radiation menopause is more delayed in its effect than surgical castration.

Relief of symptoms following the castration usually lasts for six to 24 months, the average being about nine months. The best results are noted in the case of bone metastases, and poor results are generally obtained in the presence of lung, liver, node or cerebral metastases.

Surgical castration as a prophylactic measure at mastectomy has not been carried out in any controlled series on a random basis. Both Smith and Smith (1955), and also Horsley (1951) have compared two series of Stage II cases of breast cancer treated by mastectomy

with or without surgical castration. The former showed improvement in the five-year survival from 30% to 74%, and the latter from 39% to 63% by the addition of castration. Treves (1957) showed improvement from 77% to 91% in Stage I, and from 39% to 76%, of five-year survivals in Stage II cases. With regard to X-ray menopause, Paterson (1959) carried out the only random series to our knowledge, and assessed the benefit of radiation menopause as a prophylactic measure after mastectomy. He reported that the five-year survival is significantly better for the patients who had ovarian irradiation, both in Stage I and in Stage II.

There would appear to be little doubt, therefore, as to the improvement in five-year survival following either a surgical or radiotherapeutic prophylactic castration in both Stage I and Stage II. However, particularly in the case of the patient aged under 40 years, there are often religious, social, psychological or legal reasons for not carrying out this policy in Stage I cases. The procedure is essentially not curative and its value will be manifest only in the hormone-sensitive section of those patients not cured by mastectomy. Thus, in Stage I, 10 women will be castrated for the possible prolongation of life of one. Our policy is, therefore, not rigid in Stage I. The choice between surgery and irradiation for the procedure is, in our opinion, dictated by the circumstances at mastectomy, and by the wish of the patient.

Summary.

1. Radical surgery followed by post-operative radiotherapy remains the treatment of choice for Stage I and Stage II breast cancer.

2. Radiotherapy should be based on the necessity for aiming at a critical radical dose level. This places limits on the size of the irradiated fields, and as a consequence requires their deliberate selection.

3. The criteria for the performance of radical mastectomy should be stricter than they have been in the past.

4. Exposure of the apex of the axilla and its inspection are suggested as a simple preliminary to radical mastectomy in doubtful cases.

5. Urban's extended radical mastectomy should receive serious consideration in cases of early medial quadrant carcinoma in young subjects.

6. Simple mastectomy has no place in the treatment of potentially curable carcinoma, but is often a useful palliative operation for slowly-growing malignant tumours in elderly patients.

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THE PALLIATIVE TREATMENT OF CARCINOMA OF THE BREAST.[1]

By VICTOR STONE, F.R.C.S., F.R.A.C.S.,
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THE palliative treatment of advanced carcinoma of the breast covers a wide field. This paper attempts to review the various types of treatment and to assess their value, and is based on a personal experience of more than 700 patients. It contains a plea for a definite plan of management.

Palliative treatment, to be regarded as satisfactory, should lengthen the survival time of the patient whilst maintaining her in a relative degree of comfort and giving her freedom from pain. Palliative treatment does not mean only terminal care—far from it. The patient may have years of useful life ahead of her, most of it spent preferably in the comfort of her home while she has the benefits of the treatments outlined herein.

Advances in knowledge concerning the hormonal influences on the control of cancer of the breast have brought additional factors into treatment. These consist of the administration of hormones and ablative operations on the endocrine glands. Unfortunately our knowledge is very incomplete, and consequently one sees patients submitted to one operation after another, often with disappointing results.

There is a need for some definite plan of management based on a clinical assessment. Investigations should be made that will help to indicate, as far as possible, the line of treatment likely to do the greatest amount of good and, perhaps I should add, the least amount of harm. Not until we have a better knowledge of the effects of certain operations affecting hormonal environment, and the relation of the latter to growth control, shall we be able to recommend any particular operation with confidence, and until then great caution should be exercised.

The ideal arrangement is for the patient to be admitted to hospital as soon as possible after recurrence has been detected, for a clinical assessment and a decision on a line of treatment. Once treatment has commenced the patient should be able to spend much time at home, in between any necessary periods in hospital.

CLINICAL ASSESSMENT.

The clinical assessment includes the following points:

1. The General Condition:
   - Routine general examination.
   - State of general nutrition, and cardio-vascular and respiratory systems.
   - Blood examination.
   - Urine examination, especially for albumin, sugar, ketones and bile.
2. The Extent of Local Recurrence or Metastases:
   - Local recurrence: extent and nature (e.g., permeation); determination of pathological type by biopsy of any accessible growth.
   - Metastases: lymphatic fields; opposite breast; abdomen and pelvis; determination of extent of intrathoracic and bony metastases by radiological examination of chest and skeleton.
3. Hormonal Investigations:
   - Age and menstrual history.
   - Biochemical assays of blood and urine.
   - Calcium excretion tests (if bony metastases present).
   - Cytological examination of the vagina (for œstrogen status).
   - Clinical trials of hormonal treatment.

[1] Read at a symposium on breast cancer, arranged by the Melbourne Medical Post-Graduate Committee in conjunction with the Anti-Cancer Council of Victoria, and held at the University of Melbourne on July 25, 1959.

TREATMENT.

The treatment includes (i) general supportive treatment and (ii) treatment of local recurrence and metastases.

General Supportive Treatment.

I am convinced that in some unknown manner the patient may develop a temporary resistance to the further dissemination of growth, sometimes for quite a long period, if everything possible is done to improve her general condition. This fact should be carefully considered before the patient is subjected to any drastic surgical treatment.

The patient should receive a diet of high calorie and high vitamin content. Blood restoration should be effected if there is any anæmia, and she should be given relief from pain. The withholding of blood from a patient with cancer and gross anæmia is quite unwarranted, especially if no vigorous line of treatment has as yet been commenced.

Attempts should be made to remove any sepsis and treat any intercurrent disease. Physiotherapy is most useful, and the deep-breathing exercises help to prevent lung infections. Distress caused by pleural effusion from metastases should be relieved by aspiration of fluid. After this, the injection of T.E.M. into the pleural cavity will slow down the rate of formation of further fluid. Pain must be relieved in all patients; failure to relieve pain is generally a reflection on the methods employed. Details of a plan found to be successful are given below.

Treatment of Local Recurrence.

A fungating, ulcerating tumour, either from an untreated primary growth or from recurrence of growth on the chest wall, should be treated with the object of getting intact skin over the area. The improvement in the patient's general condition as a result of the removal of the sepsis is often quite marked. It is wrong to deny the patient this benefit even if she has multiple distant secondary deposits, provided her general condition is reasonably good. The closing-over of skin ulceration can often be achieved by radiotherapy alone, or by radiotherapy combined with endocrine treatment; but if these measures fail, palliative excision should follow, provided that the area involved is not too large. As a rule there is no difficulty with healing after either local excision or mastectomy subsequent to radiotherapy. When successful, the replacement of an infected, often malodorous area by a clean scar adds greatly to the comfort of the patient (and also of those nursing her).

Treatment of Distant Metastases.

Treatment of distant metastases is by radiotherapy, hormonal control and chemotherapy.

Radiotherapy is the best method of controlling metastases confined to one or only a few localized areas, such as the long bones or the vertebræ. It is often used combined with other methods.

For widespread metastases, hormonal control should be attempted. It is accepted that approximately half the cancers of the breast are endocrine-dependent, needing an environment of either male or female sex hormone in order to grow. If the opposite sex hormone is given there will sometimes be a varying amount of retardation of the growth, either primary or secondary. Conversely, if the growth-stimulating hormone is given, the malignant tumour will grow faster, and it is sometimes alarming to see the greatly accelerated rate of growth of both primary and secondary tumours under those circumstances.

Unfortunately, there is no certain method of knowing which is the correct hormone to use, or whether any hormone will be effective.

We have found calcium excretion tests of value when bony metastases are present, but they are not infallible. The simplest method is a trial of the hormone most likely to be successful, and a quick change if there is any adverse clinical response. As a general principle, we have found the following a useful guide. Patients aged under 55 years usually do best with testosterone, particularly as regards secondary deposits in bone. Patients aged over 60 years usually do best with one of the œstrogens, particularly if the secondary deposits are in soft tissue. But there are exceptions; about one patient in 10 has a reverse response to the one expected. Recent work on œstrogen assays of urine and on vaginal cytology will probably be of value in hormonal control.

Dosages of Sex Hormones.

Androgens.—Except for testing, when testosterone propionate is used because of its quicker action, we prefer to use testosterone œnanthate ("Primotestin Depot") in a dosage of 250 mg. given intramuscularly once every 10 days. This period can be shortened to once every seven days. We have not found the use of implants very satisfactory. The response to androgens is generally fairly rapid, and side effects, although disturbing to the patients, do not cause much serious trouble. In my experience, masculinization effects on voice and growth of facial hair are nearly always present in patients who respond. I have not had the same success with preparations that do not cause these side effects. The patient can sometimes be kept under control for up to two years, and the average period is nine to 12 months. Androgens may be of benefit at any age, with great improvement in the patient's general condition and relief from pain in the bony metastases. This clinical improvement is often accompanied by definite X-ray evidence of recalcification of bone. Androgens are particularly useful when combined with X-ray therapy in the prevention of pathological fractures in long bones, and as a prophylaxis against paraplegia due to collapse of affected vertebræ.

Œstrogens.—By an arrangement with the British Empire Cancer Campaign, the Austin Hospital agreed to use for clinical trials one œstrogen only. Diethyl-stilbœstrol was chosen, and we have had an experience of some hundreds of cases. I doubt if there is any superior œstrogen for this purpose, and many of the so-called side effects have another explanation. The dose is built up gradually to 5 mg. three times a day, and seldom does one have to go above a total of 20 mg. per day. Toxic effects, if they are going to occur, usually appear in a few days. The treatment, if successful, can sometimes be carried on for years. One patient has remained well for over seven years, and the average is about two and a half years. We have experienced some unusual phenomena in sex hormone treatment. A patient may remain under control for a year or more with stilbœstrol and then go out of control, and, curiously, can be brought back under control by a change to the opposite hormone or even by the addition of the opposite hormone. I have no explanation for this, but have seen it occur on several occasions. It is dangerous to give œstrogens to patients with breast cancer aged under 55 years, except on those very rare occasions when they are indicated by laboratory tests. The most satisfactory response to œstrogens occurs in the elderly patient (aged over 65 years) with soft-tissue involvement.

Indirect Surgical Methods of Hormonal Control of Metastases.

Surgical methods of hormonal control of metastases include oophorectomy, adrenalectomy and hypophysectomy. They cannot be fully discussed here.

Oophorectomy (or the alternative irradiation of ovaries) is useful in the age group up to 60 years, but it should be remembered that even in this age group it may be of no value. It is usually a very helpful measure for younger patients. There is some preference in favour of surgical removal rather than X-ray treatment of ovaries, but it is not very marked.

Some brilliant results have been achieved by adrenalectomy, but I am not impressed with its value as a routine measure. At least seven out of 10 patients selected as probably suitable will get no benefit whatever, and I am of the opinion that in some of these there is an acceleration of the growth. There is no certain method of determining beforehand which patients will be benefited by operation. In some successful cases the improvement has lasted only

a few months, and sudden death from trivial causes has occurred rather frequently.

Struthers (1956) has shown that adrenalectomy after oophorectomy did not alter the œstrogen level in a number of post-menopausal women. This would indicate that one should not expect too much benefit from adrenalectomy in this group. Perhaps the best results are found in cases in which secondary deposits in bone are present, when there has been a temporary response to endocrines, and later the response has been lost. However, an impartial review has caused me to regard the results of adrenalectomy as disappointing.

A state loosely described as "medical adrenalectomy" can be produced by giving large doses of cortisone orally. Some rather striking improvements have been effected by this simple procedure, without the disadvantages of surgical adrenalectomy. If satisfactory, the cortisone administration can be kept on for a considerable time; but careful control of the patient is necessary, and the usual hazards of cortisone administration have to be accepted. One wonders whether the beneficial effects sometimes following adrenalectomy may not be due mainly to the necessary administration of cortisone.

I have had very little experience of the results of hypophysectomy; it has been used only in a few advanced cases. However, I believe that it may supplant adrenalectomy, because the recorded results are better. In our experience, hypophysectomy has been of no use after adrenalectomy.

Other Chemotherapeutic and Combined Methods.

Cytotoxic agents such as "Nitromin", thiotepa and lithium carbonate have been tested, with no uniform success but some striking improvement occurred in isolated cases. Thiotepa combined with androgens gave great improvement lasting several months in a few cases, and the clinical trials are being continued. It is interesting to note that the combination of two drugs or two methods of treatment sometimes appears to cause a synergistic action, with proportionately greater benefit.

Relief of Pain.

Pain not relieved by any of the foregoing treatments can be relieved by medical means in almost all cases if a definite plan is followed. Surgical methods of relieving pain, including cordotomy, have proved unsatisfactory in our cases.

At the Austin Hospital, thanks to the valuable help of Professor Frank Shaw, of the University of Melbourne, we have developed a routine which seldom fails. The basis of the plan is to combine the regular administration of morphine with a drug which is able to counteract the depressive action of morphine on the respiratory centres. The morphine antagonist we have found most useful is "Daptazole" (DAPT) and we are also getting promising results with "Tacrine" (THA). Regular six-hourly injections of morphine are given in a dose sufficient to relieve the pain. This is covered by the concurrent administration of "Daptazole" in a dose that counteracts the effects of morphine on the respiration. This drug "Daptazole" also has a marked euphoric effect, which is of help to the patient and eases the burden on those entrusted with the responsibility of looking after her.

One can give quite high doses of morphine with safety. It is amazing to see patients receiving doses of morphine up to two grains every six hours (along with appropriate dose of "Daptazole") who are able to walk about or rest in comfort, alert and free from pain. This factor alone has resulted in an almost revolutionary, and certainly very satisfactory, improvement in the terminal care of the patient.

Summary.

1. A plea is made for a new concept in the control of patients with advanced carcinoma of the breast. A plan that allows the patient long periods at home with short terms in hospital should be devised.

2. The plan commences with a clinical assessment of the patient's general condition and the extent of local recurrence or metastases, and hormonal investigations.

3. The importance of general supportive treatment is stressed.

4. Methods of control of the local recurrence and metastases are discussed.

5. Relief of pain should be achieved by following a definite medical plan.

Acknowledgements.

I wish to record my thanks to Dr. R. Kaye Scott and Professor Frank Shaw for valuable assistance in the treatment of these patients, and also to Dr. W. Moon, Medical Superintendent of the Austin Hospital, and the Records Department of this hospital for valuable help in reviewing the results of treatment.

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OCCUPATIONAL DISEASES OF THE CHEST EXCLUDING SILICOSIS.[1]

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Chief Medical Officer, Manufacturers' Mutual Insurance Ltd., Sydney.

IN DISCUSSING occupational diseases of the chest (silicosis excluded), an attempt will be made to give a broad classification of these diseases, and five main groups will be described. The grouping is not easy, as there is some overlapping in a few cases. A more detailed classification was given in a paper read at the Twelfth International Congress on Occupational Diseases at Helsinki in 1957 (Marchand, 1957). The subject is so vast that in the time available it will not be possible to do more than just mention many of these conditions.

Dusts Causing Pulmonary Fibrosis.

The first large group consists of dusts causing pulmonary fibrosis or X-ray signs simulating fibrosis. Silica is the most important of these, but it is not under discussion tonight. It is mentioned only to complete the classification. Some dusts have a high silica content, such as flint, sand, quartz, sandstone, tripoli, diatomaceous earth, etc. Others are mixed dusts, such as granite, slate, feldspar and mine dusts of many kinds.

Coal-mining dusts warrant a special heading. They may cause anthracosis, anthraco-silicosis, or in some cases mainly silicosis, as in rock-drilling and tunnelling.

Asbestos can cause a serious form of fibrosis called asbestosis, of which I have seen about 15 to 20 cases in various stages. I understand that Dr. Joseph will deal in some detail with the last two conditions.

Talcosis is not very common, but some cases have been described. Alivisatos *et alii* (1955), quoted about 30 published papers on talcosis, and described six cases in which symptoms and X-ray signs developed after less than two years' exposure.

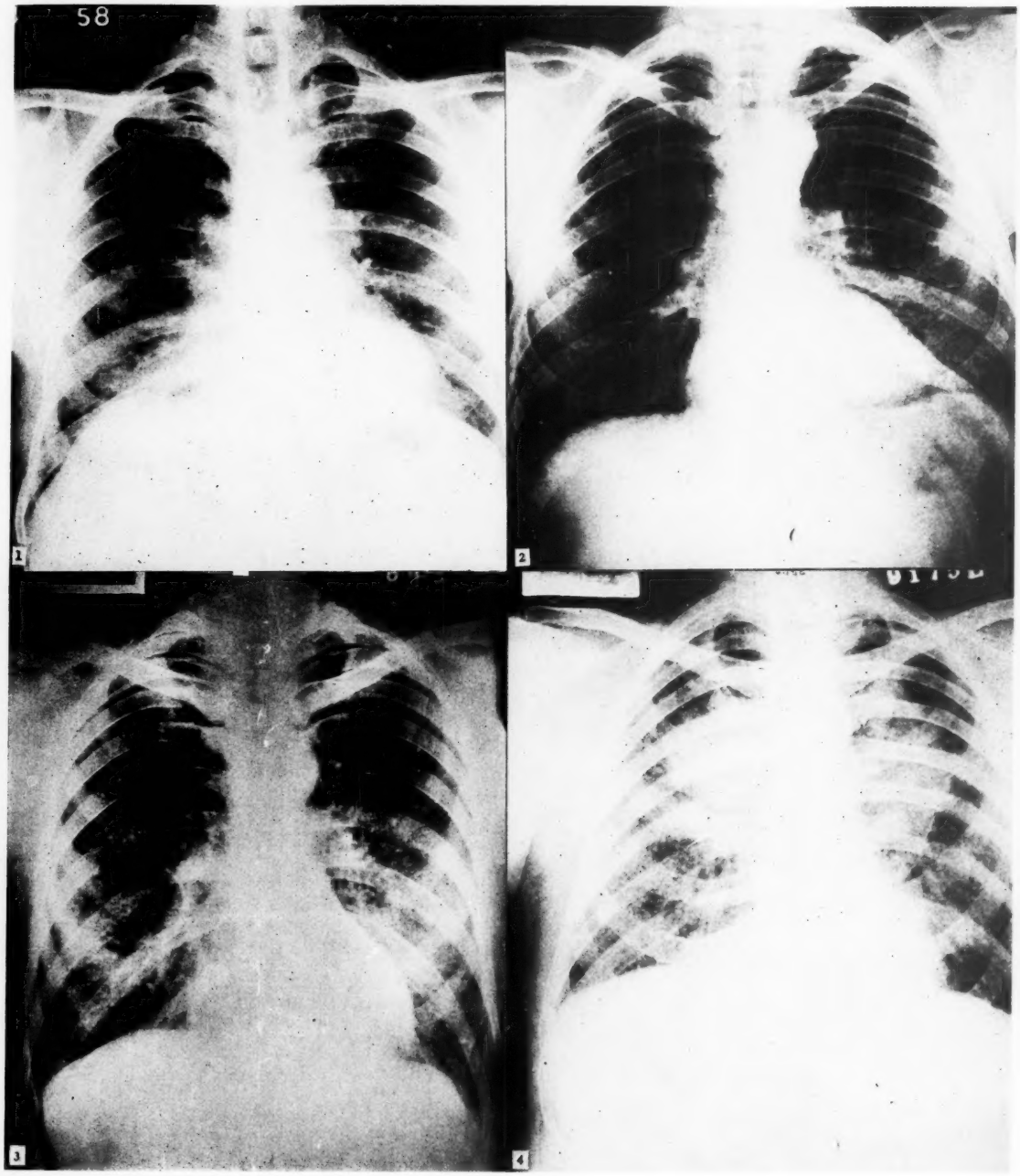
Mica pneumonoconiosis has been described, apparently not due to free silica (Dreessen *et alii*, 1940).

Kaolin or china clay used to be regarded as harmless; but Hale *et alii* (1956) have described several cases, in which it has produced disease and quoted other references. The disease, if it develops, takes many years to do so.

Cement is usually regarded as relatively harmless; but there have been a few reports in the literature of pul-

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on July 30, 1959.

ILLUSTRATIONS TO THE ARTICLE BY MAURICE JOSEPH.



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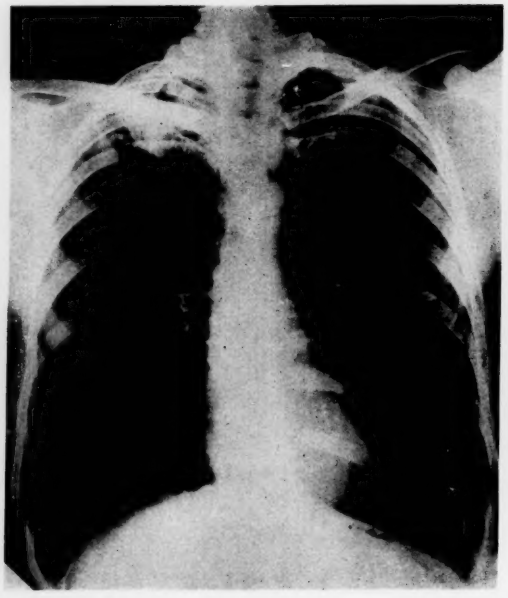


FIGURE V.

FIGURE VI.

ILLUSTRATION TO THE ARTICLE BY W. P. HOLMAN, T. H. ACKLAND AND B. A. STOLL.

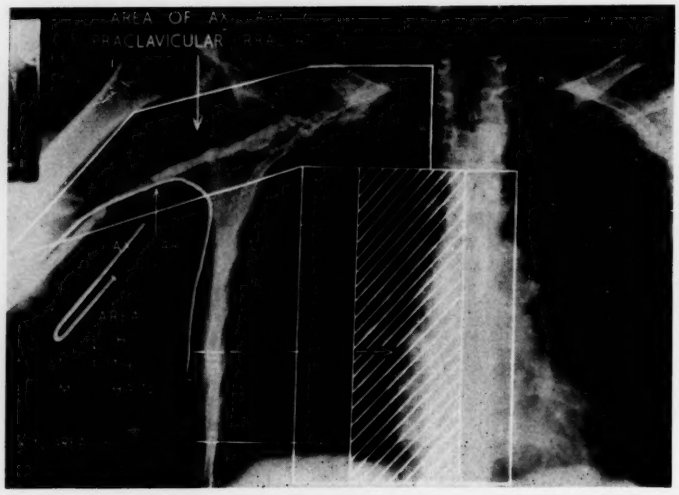


FIGURE V.

Venogram and superimposed planning of fields for irradiation of supraclavicular-axillary chain of nodes. Planning of oblique fields for irradiation of internal mammary chain of nodes.

ILLUSTRATIONS TO THE ARTICLE BY N. A. MYERS.

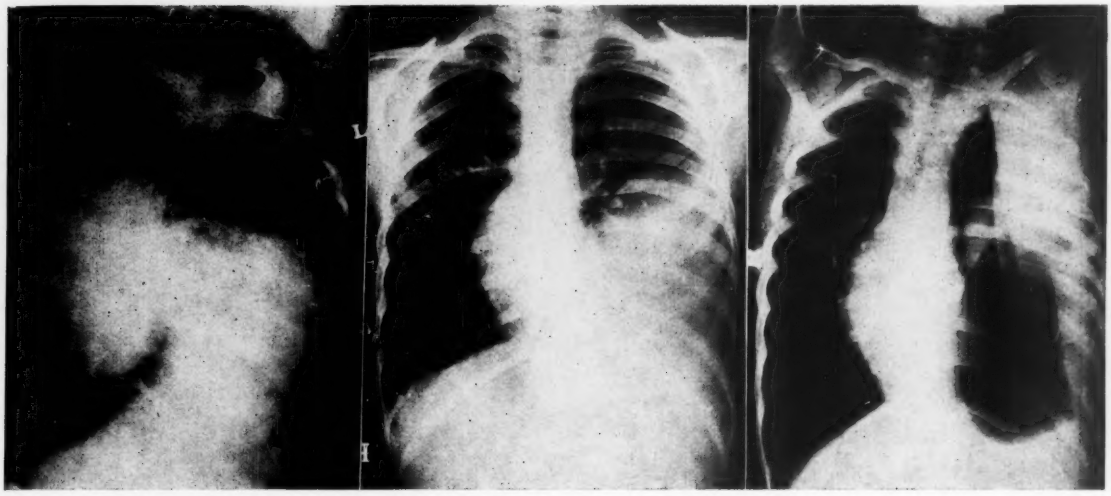


FIGURE I.—X-ray film of chest, showing huge bilateral hydatid cysts in a boy, aged 10 years, presenting with dyspnœa. FIGURE II.—X-ray film of chest: hydatid cyst of the lower lobe of the left lung, misdiagnosed as encysted empyema. FIGURE III.—Rupture of hydatid cyst of the lower lobe of the left lung resulting from paracentesis. (Same case as in Figure II: X-ray picture taken with patient lying on left side.)

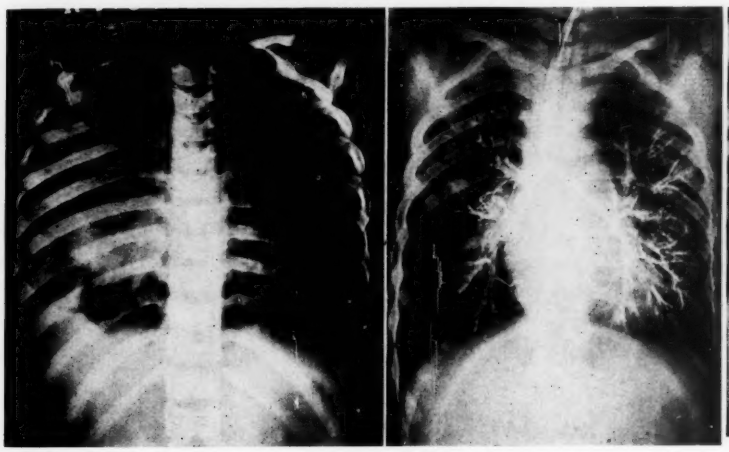


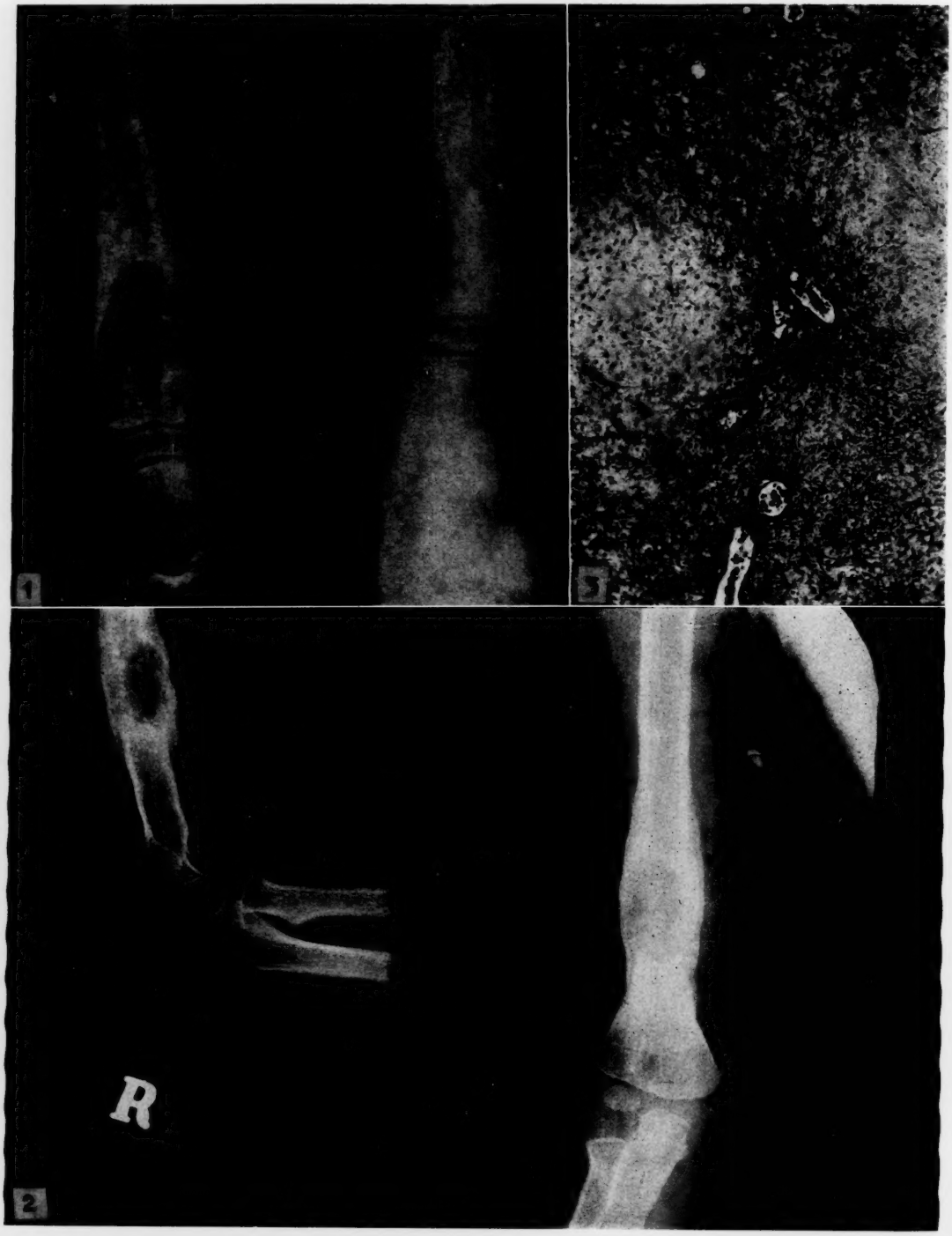
FIGURE IV.

Pre-operative X-ray film and post-operative bronchogram, the latter showing minimal residual bronchiectasis.

FIGURE V.

X-ray film of chest, showing intrapleural rupture of hydatid cyst of left lung with resultant pyopneumothorax.

ILLUSTRATIONS TO "A CONFERENCE AT SYDNEY HOSPITAL".



monary fibrosis due to this dust. The finished product contains very little free silica, which is present in somewhat greater amounts in the manufacturing process.

Aluminium has been the subject of contradictory reports. It has even been inhaled with the object of trying to prevent or cure silicosis, without much success, but without harm. However, some definite cases of aluminosis have been described, mainly in Germany, after as little as three months' exposure. In Canada, cases have been described resulting from exposure to the dust of an artificial abrasive called corundum, which is aluminium oxide.

Graphite and soot can cause a form of pneumoconiosis somewhat similar to that seen in coal miners (Meiklejohn, 1956).

Carborundum (silicon carbide) has been regarded as a safe abrasive; but two reports quoted by Donald Hunter (1955) suggest that the "pathogenicity of this substance will need to be reconsidered".

Iron oxide dust or fume (for example, from welding, silver polishing, steel grinding, etc.), and also tin and barium dusts, form deposits in the lungs and produce an X-ray picture simulating silicosis; but there is no fibrosis, and there are few symptoms, if any.

Substances Causing Acute Bronchitis, Pneumonitis or Pulmonary Œdema.

The next large group consists of those substances which can cause acute bronchitis, pneumonitis or pulmonary œdema.

Some irritant gases, such as chlorine, phosgene, fluorine, sulphur dioxide and ammonia, scarcely need description. Other chemicals which can have an acute effect on the lungs are methyl bromide, nickel carbonyl, sulphur dust, epichlorhydrin, dimethyl sulphate, perchloroethylene and isophorone. Formaldehyde is irritating to the nose and throat, but rarely causes pneumonitis. There are, no doubt, many other chemicals which could have a similar effect.

Nitrous gases can be formed by the accidental spilling of fuming nitric acid, and by the combustion of some nitrated compounds, such as celluloid. One such fire in the United States of America resulted in over 100 deaths from the inhalation of nitrous fumes. The commonest source in industry is in oxywelding or electric welding. It says much for the care taken in industry that I have not seen a case of pneumonitis definitely due to nitrous fumes. There has been an occasional claim which did not stand up to investigation. These fumes can be treacherous, because there is little or no odour at the lower dangerous concentrations, and no immediate irritation is noticed. Symptoms are usually delayed for a period of up to 24 hours, and death can occur very rapidly from acute pulmonary œdema.

Ozone is another product of electric welding, and until comparatively recently it was not thought to be harmful; but it has since been proved to cause acute pneumonitis. It is such a powerful irritant that the maximum safe concentration has been put at a figure varying from 0·1 to 1·0 p.p.m. It is rather amusing to recall that seaside resorts once advertised the beneficial qualities of this gas, and in fact expensive apparatus for its production has been manufactured and sold to a gullible public.

Manganese is a systemic poison, which causes a nervous disorder somewhat similar to Parkinson's disease; but it can also cause acute pneumonitis. It is thought that the latter is likely to occur when the particles inhaled are very small (Lloyd, Davies and Harding, 1949).

Beryllium is well known as a cause of acute pneumonitis, or of a chronic or delayed type of lung lesion in the nature of granulomatosis.

Vanadium, osmium, cadmium, nickel and selenium are also well-known irritants of the respiratory tract.

Metal-fume fever could be included conveniently in this group, though actual lung changes rarely occur. It is caused by inhaling the freshly formed oxides of many metals, the most important being zinc. A common method of acquiring this condition is by welding galvanized metals, especially in an enclosed space. Some degree of short-lived immunity seems to be developed, so that is is more common on Mondays, after the week-end away from work. It is an acute illness with a high fever, and full recovery occurs in 24 to 48 hours.

Accidental aspiration of some industrial hydrocarbons, such as petrol and kerosene (for example, when using mouth suction to get a siphon started), causes a very acute chemical pneumonitis, similar to that caused in children who are poisoned by drinking kerosene, which is more dangerous when aspirated into the lungs than when ingested. Severe poisoning by inhalation of petrol vapour is sometimes followed by pneumonitis.

Materials Causing Allergic Reactions.

Another important group consists of those materials which may cause allergic reactions. Some of these are of vegetable origin, such as cotton, bagasse, flax, hemp, jute, grain, seeds, pollens, derris, flour, hay, straw, many types of wood, tea, tobacco, sisal, grasses, nuts, cork, malt, linseed, spices, gum arabic, gum acacia, gum tragacanth and tamarind. Some are of animal origin, such as hair, feathers, fur, bone, horn, ivory, leather, silk and wool. Others again are chemicals, as, for example, paraphenylene diamine (used as a fur dye), isocyanates, ipecacuanha, platinum salts, sulphaguanidine, epichlorhydrin, etc. In fact, most of the common extrinsic causes of asthma can be met with industrially. Time will not permit any detailed discussion of these; but cotton deserves a mention as the cause of byssinosis, though it is not encountered frequently in Australia. This usually takes at least 10 years to develop, and often 20 or 30 years. At first asthmatic symptoms occur only on Mondays, and as the condition becomes worse it spreads over to other days of the week, but is worst on Mondays. Chronic bronchitis may or may not develop as well in the later stages. With some of the chemicals sensitization can become extreme, as in a case of asthma due to isocyanates (which are used in making "Estafoam" and certain types of lacquers). This man developed such an acute sensitivity that contact outside the factory with a man who had just been working with this material brought on an acute attack.

Infections or Infestations.

Many infections or infestations can be occupational in origin, or can be influenced by working conditions.

I am indebted to Dr. Cotter Harvey for some of these notes about pulmonary tuberculosis, which is related to occupation under the following conditions: (*a*) if there is occupational contact (as in the case of nurses, doctors, laboratory and hospital attendants, etc.); (*b*) if silicosis is present, as that seems to be a strong predisposing factor; (*c*) in some cases of direct trauma, when the evidence of the nature and site of the injury is clear-cut, the fresh symptoms become apparent in four to six weeks, and the X-ray picture shows fresh disease on old, or fresh activity; (*d*) if the working conditions are extreme and such as to cause debilitation and unusual fatigue.

"Q" fever is often associated with atypical pneumonia. This disease can be milk-borne, but is often occupational in origin, and I have dealt with a good many claims from abattoir workers in northern New South Wales. A recent report by Marmion and Stoker (1958) suggests that, at least in some districts in England, it occurs in a considerable proportion of people not directly associated with animals; but no comparable investigations have been carried out in Australia, and until that has been done I believe that I have no option but to accept such claims if the diagnosis is beyond doubt and if there has been potential occupational contact. It is either becoming more common, or is being recognized more frequently.

Leptospirosis and brucellosis occur in Australia under similar occupational conditions to those which cause "Q"

fever, but are less often associated with pulmonary complications.

Other less common infections which can affect the lungs are psittacosis, anthrax, glanders, tularæmia, and various fungous diseases such as aspergillosis, actinomycosis, moniliasis, coccidioidosis and histoplasmosis.

It is theoretically possible that some infestations, such as hydatid disease and bilharziasis, could be occupational in origin, and in the case of a hydatid cyst direct injury could conceivably cause some aggravation. I have dealt with only one claim for causation of hydatid disease, and as the man was a slaughterman and claimed that he acquired the disease directly from a sheep, I could not admit the claim.

Substances Thought to Cause Lung Cancers.

The final group in the classification which I have adopted consists of the substances causing, or thought to cause, lung cancers. There are a few in which the relationship seems to be fairly definite, and some others in which there is considerable doubt. Lung cancers constitute about 10% of occupational cancers.

Nickel.

A considerably increased incidence of cancer of the nose and lung was found in nickel refinery workers in South Wales. At first this was thought to be due to the gas, nickel carbonyl; but the latest view of Morgan, who is chief medical officer for the factory which does most of the nickel refining in Great Britain, is that the carcinogen is contained in the heated calcined dusts (Morgan, 1958). He even thought it might have been the arsenic content of the dust that was responsible.

Chromates.

Various authors have reported detailed studies which leave little room for doubt that chromate workers are more liable than others to develop lung cancer; but the actual carcinogenic factor has not been identified.

Radioactive Substances.

Radioactive substances, as would be expected, have also been incriminated as a cause of lung cancer. It has been known since the sixteenth century that miners in Schneeberg, Saxony, were likely to die from a lung condition which was shown about 1879 to be a malignant growth. More recently a similar incidence of lung cancer has been recognized in miners in nearby Joachimsthal in Bohemia. The dust and air in these mines are radioactive, and although for a long time arsenic was considered to be the carcinogen, most observers now regard radioactivity as the cause (Hamilton and Hardy, 1949). Cancer of the lung has also been reported in radium laboratory workers.

Asbestos.

An excessive mortality from cancer of the lung in cases of asbestosis has been recorded by several observers, and this association is now accepted by most authorities.

Other Suspected Lung Carcinogens.

The manufacture of iso-propyl alcohol is said to be associated with the development of cancer of the respiratory tract, mainly the nasal sinuses and larynx, but a few cases of lung cancer have been reported.

Another interesting possibility is the association of a combination of iron and silica, causing sidero-silicosis, with cancer of the lung. In the hæmatite miners and some steel grinders and other foundry workers, a somewhat increased incidence of the disease was found; but this association must still be regarded as unproven.

Two other doubtful lung carcinogens are coal-tar fumes and arsenic. There is some evidence (Kennaway and Kennaway, 1936; Doll, 1952; and some observers in Japan and Canada) that there is a higher incidence of lung cancer in workers exposed to tarry fumes; but more recent work in England led Reid and Buck (1956) to conclude that "the mortality in the coking industry from cancer in general and cancer of the lung in particular, is not as excessive as had been feared, and may in fact be negligible". In many years' experience with the largest gas-making organization in New South Wales, I have not seen a claim for lung cancer. I did deal with one in a man who had some contact with tarry fumes; but as he smoked heavily and his contact was intermittent, he did not succeed in this part of his claim.

Arsenic has had a bad reputation for a long time as a carcinogen, both cutaneous and pulmonary, and some statistical studies favour the association of arsenic and lung cancer; but the number of workers concerned was rather small, and other studies tended to disprove the association. In a recent monograph by Eckardt (1959) on industrial carcinogens, arsenic is not mentioned as a possible cause of lung cancer. Hunter (1955) states that all the available evidence is contrary to the view that arsenic is responsible for industrial cancer in many occupations.

A great deal of work is still required before the problem of occupational carcinogenesis can approach solution.

Discussion.

This finishes the systematic survey of occupational diseases of the lungs. I should now like to conclude by mentioning three chest conditions which give rise to claims, and I shall give my views and hope that some discussion will ensue in regard to these controversial matters.

In trying to assess the relationship between a medical condition and occupational factors, there are three ways of tackling the problem: (i) epidemiological or statistical studies; (ii) theoretical considerations, (iii) experimental and operative observations. If one of these contradicts the others, the relationship is more doubtful than if they all agree.

Spontaneous pneumothorax, when it occurs at work, is mostly claimed to have been caused by exertion, and the theory usually propounded by the medical witness is that the exertion made the worker strain with a closed glottis, thus raising the intrapulmonary pressure and causing an emphysematous bleb to burst, like a blow-out in a motor-car tyre or football bladder which has a weak spot in the wall. Apart from the doubtful proposition that such mild exertion as, for example, sweeping would make it necessary for a man to hold his breath and strain, consideration should be given to the mechanics of respiration, which is different from the mechanics of inflating a football bladder. The increased intrapulmonary pressure caused by straining against a closed glottis is due to pressure on the lung by the diaphragm and chest wall, which is in contact with and supports the lung as the football casing supports the football bladder, so that any weak spot such as an emphysematous bleb is well supported. On the other hand, during a sudden deep inspiration the chest wall and diaphragm are expanded in a direction away from the lung, and if a weak spot was present during this sucking process, it would tend to burst through and cause the pneumothorax. This, of course, leaves out the valvular theory, in which a local build-up of pressure in the bleb is thought to occur even during quiet respiration, and could account for cases in which pneumothorax occurs at rest or during sleep. I have found only three references quoting statistics regarding the activity of the patient at the time of onset of the pneumothorax. An article by Moxon (1950) mentions 26 cases, the activity at onset being known in 19. Of these patients, one had just jumped aboard a bus, three were walking, five were standing, three were sitting, one was bending, four were lying down, and two were asleep. Corsello (1949) makes the following statement:

> Strenuous physical activity is not a necessary prerequisite for pneumothorax to happen. Leach, in his report of 126 cases, noted that 48 per cent. of the

patients were at rest when the pneumothorax occurred and 38 per cent. were engaged in mild activity. Schneider and Reissman found that only 30 of their 100 cases developed the pneumothorax during strenuous activity.

Another hardy perennial in the way of claims is one for pneumonia, allegedly precipitated by exposure to cold or by getting wet. I can recall in my youth over 50 years ago, in a hot climate out west, not being allowed to sit between two open doorways because it was "in a draught", even if one could not feel the slightest movement of the air. This idea still seems to persist in some medical minds. It is perpetuated in the name given to an acute coryza—when one is inflicted with that, one has "caught a cold". It is common knowledge, though as far as I know there is no statistical evidence about it, that innumerable soldiers, sailors, fishermen, golfers, etc., get cold and wet without any harmful sequels. Hundreds of thousands of people spend the day at the beach, baking in the hot sun, and then spending an hour or more in the comparatively cold water, and then repeating the process. Certain operations are done after the induction of hypothermia, when the patient is kept at a subnormal temperature, sometimes for many hours—and these are often poor-risk subjects. This surely does not make them liable to develop pneumona because of the hypothermia, or this disadvantage would outweigh the advantages. Some experimental work has been done in England and America, and it has not been shown that resistance to the common cold can be lowered by chilling, wearing wet socks, standing in draughts, etc. (Andrewes, 1958; Leading Article, 1957). Most of us have had many colds in our lifetime. How often has such a condition been precipitated by any extrinsic factor of which we are aware?

The last condition which I wish to mention is the relationship of chronic bronchitis to dusty conditions. I would certainly not be so foolish as to state that there is never any such relationship; but I do feel that it is too easy to say that if a man has been exposed to some unspecified amount of dust, it must be the cause or a main contributory factor in the development of his chronic bronchitis. The relationship is not as simple and certain as that. It is a subject which has been found difficult to investigate for several reasons, such as the uncertainty of the diagnosis of chronic bronchitis, and the influence of other factors, such as smoking, chemical atmospheric pollution, change of occupation, social class, allergy, infections, and the effects of advancing years. Another difficulty is that most of us try to look for a cause of our own illnesses, and a chronic bronchitic is more likely to complain of dustiness, draughts, extremes of temperature, etc., when asked about these things, than is a normal person on whom such working conditions have no obvious effect. Time will not permit a long discussion on this subject; but I am quoting a number of articles to which reference can be made, and it will be seen that evidence is accumulating both for and against a causal relationship between chronic bronchitis and dusty working conditions (Pemberton and Goldberg, 1954; Leading Article, 1956; Higgins *et alii*, 1956; Pattle and Cullumbine, 1956; Gregory, 1955; Higgins, 1957; Leading Article, 1959; Higgins and Cochran, 1958; N.A.P.T., 1957; Ogilvie and Newell, 1957; Stuart-Harris and Hanley, 1957). In a personal communication from Dr. I. T. T. Higgins, of the Medical Research Council Pneumonoconiosis Research Unit, he makes the following statement:

> I think it would be correct to say that this problem is by no means as straightforward as one might conclude from reading text books on bronchitis. We ourselves have not found a very clear relationship between the indirect measure of dust exposure that we have used in our survey work and the prevalence of symptoms.

There is evidence in recent years of an increasing interest in chronic bronchitis, so it is hoped that before long many aspects of this disease will be clearer to us than they are now.

Conclusion.

My final plea, which applies to all industrial medical cases, is for care to be taken to get a complete detailed history, which may even necessitate a visit or a telephone call to the factory. In particular, the exact time relationship of the onset of the medical condition to the incident or the circumstances at work should be sought. Too often one reads that a certain condition "followed" a particular incident. That is not enough information. Another common history seen is that a man lifted a weight or pulled a lever and felt a pain. Closer inquiry may elicit the information that the pain came two hours or two days after the incident. Getting the story of the sequence of events in exact chronological order is one of the most important parts of the history in industrial medicine.

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OCCUPATIONAL DISEASES OF THE CHEST, EXCLUDING SILICOSIS.[1]

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EVEN if one excludes silicosis, industrial lung disease is still a tremendous subject and far too large even to attempt to cover in one short lecture. I propose, therefore, to limit my remarks to two conditions which have come within my personal experience more frequently than other industrial lung diseases—namely, asbestosis and coal-workers' pneumonoconiosis. Also I shall deal with these from the point of view of the thoracic physician, and not that of the industrial hygienist.

The uses of asbestos have been known for over two thousand years, one of the earliest references to it being by Herodotus in 450 B.C.; he described how the Romans mined it in the Italian Alps and Urals and used it for enshrouding their corpses before cremation. The name "asbestos" is derived from the Greek meaning "inconsumable", and Plutarch referred to the wicks of the lamps of the Vestal Virgins as "asbesta". The dust associated with the handling of asbestos created difficulties in those days as at present, and Pliny in A.D. 50 referred to the use of respirators to avoid the inhalation of dust.

The Australian production of asbestos is not large; it amounted to 2500 tons in 1954, of which all but 450 tons came from Western Australia. This by no means satisfies the requirements of industry, and large quantities are imported, mainly from South Africa. It arrives in sacks as the crushed rock, and men employed in emptying these have been exposed to a high concentration of the dust. Asbestos fibres of extreme fineness can be obtained, spun into yarn and woven into cloth which has a great variety of uses. In addition to being woven, it can be ground and mixed with other materials to make insulating slabs, boarding, brake and clutch linings, electrodes for welding, acid-resisting filtering cloths, packings, jointings and wrappings for steam pipes. The dust given off in the process is mostly less than 5μ in diameter and 10μ in length. As with other dust diseases of the lungs, the occurrence of asbestosis depends upon the concentration of dust to which the worker is exposed and on the duration of exposure. There are, nevertheless, individual variations, as with silicosis, among workers apparently exposed to similar conditions.

Clinically the mode of presentation is with dyspnœa of gradually increasing intensity, often with a cough which is generally non-productive. There are usually no physical signs in the early stages of the disease, but later râles develop, with evidence of impaired respiratory function and cyanosis and clubbing of the fingers in advanced cases. As was mentioned previously, the cough is mostly non-productive, but examination of the scanty sputum produced will often reveal asbestos bodies. These highly characteristic structures are long and tapering, up to 75μ in length, and are a golden-yellow or brown colour. They tend to be segmented, and are of an elongated, dumb-bell shape. They have been shown to consist of a central core of asbestos, upon which a colloidal combination of tissue protein and silica has been adsorbed. This colloidal covering is soluble in strong acids and alkalis which leave the central asbestos fibre bare. Asbestos bodies obtained in a pure state by tryptic digestion of the lungs have been found to consist of approximately 70% organic matter and 30% inorganic matter. They may be found in sputum before there is any radiological evidence of asbestosis, and must be regarded as an indication of inhalation of asbestos dust, but not necessarily of clinical asbestosis.

Radiologically the appearances are fairly characteristic. Usually there is a diffuse haziness throughout the lower lung fields generally with obliteration of the normally sharp margins of the cardiac silhouette (Figure I[1]). Occasionally there are scattered, granular and punctate mottled shadows, but these have not the well-defined margins of the coarser silicotic nodulation.

Histologically, asbestosis produces diffuse fibrosis, starting mainly around the terminal bronchioles and atria as collar-like sheaths and extending peripherally as the disease progresses; it produces profound thickening of the alveolar walls, the interlobar septa and the pleura, and throughout the lung tissue are seen fibres of asbestos either singly or in clumps, as well as the characteristic asbestos bodies. Those parts of the lung not involved in the fibrotic process naturally become emphysematous.

Dyspnœa is the outstanding symptom, and reduction of respiratory capacity will be progressive unless the patient is removed from dust exposure at a very early stage of the disease. This is readily understood, because the primary condition is pulmonary fibrosis which becomes more marked with the passage of time even if the subject is removed from contact with the dust. Pleural thickening and emphysema are natural sequelæ and add to the patient's increasing respiratory disability. He becomes liable to chronic or recurrent bronchitis, and ultimately to the development of cor pulmonale.

From a study of 25 patients with asbestosis, Hugh Jones, John Read and Roger Williams (to be published) concluded that the primary disability was a diffusion defect, as would be expected from the thickening of the alveolar walls. They also found that those patients who had much fibrosis had diminished vital capacities, but no appreciable reduction in maximum breathing capacity unless bronchitis was present.

A patient recently studied was a man, aged 47 years, with a history of exposure to asbestos dust from 1927 until 1956, when he left the industry because of increasing dyspnœa. He had worked mainly as a lagger and asbestos mattress maker with various firms, with a break of four years during which he served with the Royal Australian Air Force. He was dyspnœic on mild exertion, and had a plethoric facies and definite clubbing of the fingers. Medium crepitations were heard over the lower lobes of both lungs, and an X-ray examination of his chest showed the appearance seen in Figure II. It is interesting that the film taken three years ago (1957) was passed as normal, and it was seen from a comparison of these skiagrams that the fibrosis has mainly developed since he left the absestos industry. The respiratory function tests were carried out by Dr. John Colebatch, with the following results. The vital capacity was 3·8 litres (100%), the maximal breathing capacity was 82 litres per minute (73%), and the diffusion capacity for carbon monoxide was 10 (55% of normal).

This shows that, though there is slight airway impairment, the main defect is in the matter of diffusion across the alveolo-capillary membrane.

Last October a group of six men suffering from asbestosis was examined. They had all worked for a period of approximately ten years in what was known as the "asbestos gang", whose work involved emptying bags of imported asbestos into a crushing machine. All complained of dyspnœa and cough of varying degrees of severity. Some had no abnormal physical signs, and in others crepitations were heard at the lung bases. The X-ray appearances were characteristic, and although the men were removed from contact with asbestos dust when the disease was first discovered to be present in them in 1956, there has been progressive fibrosis. There was a fairly good correlation between the severity of symptoms and the results of limited respiratory function tests carried out, but the correlation between these and the X-ray appearances was not close.

Asbestosis is not only a crippling disease in itself, but renders the subject more susceptible to two serious diseases. The studies of Wood and Gloyne and of Middleton and McLoughlin indicate that there is an increased susceptibility to tuberculosis in patients with asbestosis, although this has been questioned by other workers. Studies by Doll, and by Lynch *et alii*, both in 1955, indicate quite definitely that carcinoma of the lung has a higher incidence in asbestosis sufferers than in the general population. This high incidence of bronchial carcinoma has

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on July 30, 1959.

[1] For Figures I-VI, see art-paper supplement.

been confirmed by others and is widely accepted. Doll's analysis has shown that the average risk among men employed in asbestos for 20 years or more has been ten times that experienced by the general population.

The second topic I wish to present is coal-workers' pneumonoconiosis, and again in the time available only certain aspects can be dealt with. The coal deposits of New South Wales are the most important and extensively worked in Australia, and are situated in the vicinity of Newcastle, Bulli and Lithgow, being referred to respectively as the northern, southern and western coalfields. Approximately 13,250 men are employed in these mines, and the extent of the pneumonoconiosis problem can be gauged by the fact that at present 870 men are receiving compensation for this condition (420 total and 450 partial). A great deal of work has been done on this subject by the Welsh National School of Medicine at Cardiff, and our knowledge of the pathology of the disease is largely based on the observations of Professor J. Gough. In our own State, pioneering work was done in this field by the late Charles Badham and carried on by Dr. W. E. George and others.

It was once thought that miners who developed pneumonoconiosis did so because of the inhalation of silica mixed with coal dust, and that the coal dust itself was inert. W. R. James has shown that the dust to which the miners of South Wales are exposed rarely contains more than 1% free silica; moreover, in 1000 cases of coal-workers' pneumonoconiosis studied by James, 19 subjects had worked as trimmers loading coal into the holds of ships and had never been underground; seven of these had confluent massive fibrosis. This accords with Gough's (1940) findings, and disposes of the theory that the condition is due to highly siliceous dust from rock strata adjacent to the coal seams. Moreover, the pathology is different from that of silicosis.

The development of pneumonoconiosis depends on two groups of factors, one relating to the dust itself and the other to the individual; given identical dust factors with regard to particle size and atmospheric concentration, individuals vary in the degree and extent of the pneumonoconiosis that develops. These factors are difficult to assess; but it appears that the efficiency of the nasal filter and the presence of other pulmonary disease, such as bronchitis, are important. The claims of metabolic and genetic factors, physiological peculiarities of the lung and its lymphatic system and physiological response to climatic factors and others have been invoked, but none appears to have been substantiated.

Dust particles greater than 10μ in diameter seldom reach the lung alveoli, either settling out of the atmosphere or being filtered off by the respiratory tract; however, particles less than 3μ can reach the alveolar spaces in large numbers. Here they are taken up by phagocytes which apparently originate in the alveolar walls, and then either are wafted upwards and finally expectorated, or enter the lymphatic vessels and are transported to the lymphoid tissue of the lungs and thence to the hilar lymph nodes, from where they may travel to the scalene nodes which are accessible to biopsy. Still other phagocytes with their carbon load gather in foci around the respiratory bronchioles, where they constitute the characteristic lesion of coal-workers' pneumonoconiosis. These lesions are stellate and intensely black, are scattered throughout the lung, and vary in diameter from microscopic dimensions to about 5 mm.; but they contain comparatively little fibrous tissue. Gough, by the use of whole-lung sections, has demonstrated these lesions beautifully, showing all gradations of simple coal-dust pneumonoconiosis.

Gough pointed out the essential difference between silicosis and coal-workers' pneumonoconiosis, and described the characteristic focal emphysema of the latter. However, it remained for A. G. Heppleston, by painstaking serial section technique, to demonstrate that the proximal limit of this sheath of dust cells is regularly in the region of the terminal bronchioles, but distally extends for a variable distance up to the division of the final order of respiratory bronchiole, of which there may be three, depending upon the degree of dust accumulation. In the course of time the ensheathed bronchioles dilate, producing what is known as focal emphysema. Until Heppleston's work it was generally considered that focal emphysema was the result of obstruction of air passages by dust fibrosis. This histological condition of focal emphysema forms the basis on which it is possible to understand the dyspnœa of simple pneumonoconiosis. As respiratory bronchioles dilate, their cross-sectional area, and therefore their volume, increases in proportion to the squares of their radii. This is equivalent to an increase in the volume of functional dead space, and leads to deficient aeration of the distally placed segments of the airway.

To add to the coal-miners' disability, generalized vesicular emphysema not infrequently develops. This is distinct from focal emphysema, and is characterized by dilatation of alveolar ducts, atria and alveolar sacs. The cause of this condition in coal-miners is the same as that in the majority of non-miners who develop emphysema—namely, chronic bronchitis. Pemberton has shown by a careful comparative study that chronic bronchitis is approximately three times as common in coal-workers as in other industrial groups. In case it could be considered that smoking was a factor, he investigated this possibility and found that a greater proportion of the coal-workers were lifelong non-smokers or had given up smoking, than in the non-mining groups studied. There was also a considerably lower proportion of heavy cigarette smokers among the coal-workers. It would be surprising if the inhalation of dust-laden atmosphere over a long period did not cause such mechanical irritation as would, in the long run, lead to the development of chronic bronchitis.

Radiologically, simple pneumonoconiosis is characterized by fine opacities described and classified in the International System as category 1, 2 and 3. In some cases a condition is superimposed which has been termed "confluent massive fibrosis". This consists of well-marked collagenous fibrosis forming masses varying in size from 0·5 to 15 cm. in diameter. They occur most commonly in the apices of the upper and lower lobes—that is, in positions most favoured by tuberculosis. Although the ætiology of this condition has been disputed for some years, there is now fairly general agreement that it results from superimposed tuberculous infection. James in 1954 was able to find histological and bacteriological evidence of tuberculosis in 40% of the massive lesions present in 245 South Wales coal-workers. Since it is difficult to believe that the bacilli penetrate into existing massive lesions, one must postulate that the organisms reached their situations by being inhaled before or with the dust. These lesions sometimes cavitate, causing the production of intensely black sputum which has been likened to printers' ink. Confluent massive fibrosis has been divided into categories A, B and C on the radiological appearances of increasing severity.

In 1953 Caplan described another form of massive fibrosis, in which the round opacities were multiple and well defined, and distributed throughout both lung fields, but particularly at the periphery. These lesions occurred in coal-miners with a mild degree of simple pneumonoconiosis who also had rheumatoid arthritis, and were shown to be histologically distinguishable from confluent massive fibrosis. The condition has been given the name of "Caplan's syndrome".

In recent years it has become increasingly recognized that severe respiratory disability can exist without radiological evidence of pneumonoconiosis. There is a category 0 in coal-workers' pneumonoconiosis; that is, significant and even disabling disease can exist with an X-ray picture in which recognizable dust opacities are either absent or sparser than in the standard category 1 film. I have seen a number of such patients from the Cessnock mines, and the details of one patient who had a lobe removed because of a suspicious apical lesion will illustrate the point.

The patient was a man, aged 54 years, who had been a miner for 40 years. He had first noticed dyspnœa three years before he was referred to me on June 19, 1958. This had reached the stage at which he said he found it difficult to walk out of the pit, and even the exertion of bathing and dressing caused breathlessness. He had only a slight cough, and the sputum he produced was white and frothy. His chest skiagram in 1956 had been passed as clear, but a current one showed an opacity at the apex of the right lung (Figure V). Tuberculosis was considered the most likely diagnosis; but repeated sputum tests gave negative results, and because of the possibility of carcinoma thoracotomy was advised. Clinically and radiologically the patient appeared to have marked emphysema, and this was confirmed by respiratory function test carried out by Dr. Colebatch. His vital capacity was normal (3·48 litres), but the maximum breathing capacity was considerably reduced, being 36·8 litres per minute (36% of the predicted value). The total lung capacity was 126% of predicted level and the residual volume 161%, giving a residual volume to total lung capacity ratio of 52. He was subjected to surgery, and although a less extensive operation would have been preferred, a right upper and middle lobectomy had to be performed because of technical difficulties. The lesion proved to be tuberculous; but the interest in the case is the fact that examination of the lung sections showed a marked degree of pneumonoconiosis, despite the absence of dust opacities in the skiagram (Figure VI).

The lack of correlation between the radiographic appearances of pneumonoconiosis and the degree of respiratory disability is also exemplified by studying the respiratory function tests of the patients whose skiagrams I used to demonstrate the radiological categories of simple pneumonoconiosis and confluent massive fibrosis. Reference to Table I will show that a man in category 3 can have

TABLE I.

Results of Respiratory Function Tests in Patients with Coal-Workers' Pneumonoconiosis.

Category.	Age. (Years.)	Years in Mine.	Vital Capacity.		Maximal Breathing Capacity.		R.V. : T.L.C.[1]
			Litres per Minute.	Percentage of Normal.	Litres per Minute.	Percentage of Normal.	
1	53	39	4·0	106	86	77	32
1	50	33	2·8	83	49	53	50
3	59	32	4·6	116	85	81	35
A	63	45	3·8	130	94	106	33
B	70	32	1·8	60	30	34	55
C	60	30	4·1	127	74	73	33

[1] Ratio of residual volume to total lung capacity.

considerably less respiratory disability than one in category 2; that virtually no respiratory disability may exist with category A, confluent massive fibrosis, and a category C man may have appreciably better respiratory function than one in category B. As long ago as 1948 Fletcher described the case of a coal-worker who was the local champion for the 75 yards sprint, and whose chest skiagram showed progressive massive fibrosis.

Conclusions.

From the foregoing, as well as from other published work which time prevents me from including in this paper, the following conclusions must be drawn.

1. The respiratory disability of coal-workers is primarily an airway disease, and is due to (*a*) focal emphysema and (*b*) chronic bronchitis and generalized vesicular emphysema.

2. There is no correlation between respiratory disability and the X-ray appearances in the chest.

3. In the matter of determining compensation, the X-ray findings in the chest may be misleading and unjust in two directions: (*a*) a worker with radiologically evident pneumonoconiosis may have no respiratory disability; (*b*) a worker with severe disability due to his occupation may have no radiological evidence of pneumonoconiosis.

4. Tests of respiratory function such as that of the maximal breathing capacity and timed vital capacity, in conjunction with clinical evidence of respiratory disability, provide a better indication of the state of the worker's pulmonary health than an X-ray examination, and should supplement the latter in the regular assessment of the miner.

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HYDATID DISEASE IN A CHILDREN'S HOSPITAL.

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SINCE 1938, 72 patients with hydatid disease have been admitted to the Royal Children's Hospital, Melbourne. Eleven of these were seen for the first time in 1957. The continued prevalence of hydatid disease provided the stimulus for a review of the pædiatric aspects of the condition. Information obtained from other centres in Australia indicates that the problem is by no means purely local, because over 1000 patients with hydatid disease have been admitted to public hospitals in the capital cities of Australia during the past twenty years. A further 86 patients with pulmonary hydatid disease have been treated by Dr. S. C. Fitzpatrick of Hamilton, Victoria, since 1920.

Clinical Material.

Sixty-seven of the patients were Victorian, three were referred from Tasmania and two came from south-western New South Wales. Thirty-five of the Victorian patients were living in Melbourne at the time of their admission to hospital. Some had previously lived, or spent holiday

periods, in the country. The total bed occupancy was 5370 days, each patient averaging 74 days in hospital. Four deaths occurred, all within the first half of the 20-year period. There were 50 boys and 22 girls in this series, and their ages ranged from 2 to 13 years. Ten children were aged three years when the diagnosis was made.

Classification of the cases according to the distribution of the disease enabled the following four groups to be recognized:

Group I, pulmonary involvement alone: 36 cases.

Group II, hepatic involvement alone: 18 cases.

Group III, pulmonary and hepatic involvement: nine cases.

Group IV, miscellaneous involvement: nine cases.

In the fourth group, in addition to the lungs and liver, organs involved included the spleen, kidney and brain. In one case a cyst was present in the abdominal wall, and in four cases the peritoneal cavity was affected.

In the 72 cases, 173 cysts were identified, distributed throughout the body as follows: lungs, 82 cysts; liver, 75 cysts; peritoneum, nine cysts; spleen, three cysts; brain, two cysts; kidney, one cyst; abdominal wall, one cyst.

The 82 pulmonary cysts were distributed as follows: (i) Right lung: 38 cysts; upper lobe, eight; middle lobe, three; lower lobe, 20; unspecified, seven. (ii) Left lung: 41 cysts; upper lobe, 14; lower lobe, 15; unspecified, 12. (iii) Pleural cavity: One cyst. (iv) Unspecified: Two cysts.

Method of Presentation.

Forty-one patients presented with pulmonary features; 19 of these had acute symptoms, and 19 had chronic symptoms. In three symptomless cases the diagnosis was made after routine radiological examination.

Twenty-nine patients presented with abdominal symptoms. Of the remaining two patients (both with cerebral involvement), one presented with asthenia and the other with drowsiness. Details regarding the specific presenting symptoms are set out in Table I.

Investigation.

Radiological examination was carried out in all cases. Serological tests were performed in 63 cases. In 48 both the Casoni test and the hydatid complement fixation test were performed; in eight cases the Casoni test alone was performed, in seven the hydatid complement fixation test alone. The results of these tests are detailed in Table II.

Forty-six estimations of the number of circulating eosinophils were available. Sixteen of these revealed eosinophil counts less than 300 per cubic millimetre; in the other 30 the counts ranged from 300 to 6000 per cubic millimetre.

Discussion.

The main purpose of this paper is to present the clinical features of hydatid disease in children. In this series 70% of the cases occurred in boys, and 76% were aged between 3 and 10 years when the diagnosis was made. In view of our knowledge of the life cycle of the parasite and the method of human infestation, it would seem that the play habits of boys make them more vulnerable than girls, and, at the same time hands may not be thoroughly washed in these early years of childhood. As a toddler, the child's hands are washed by his mother; in the second decade of life the visit to the bathroom may provide more infection.

The situation is very well put by Sir Harold Dew in the following statement:

> The frequency of infestation in the child is explained by the ignorance of hygienic matters, his habit of putting things into his mouth, his plantigrade habits, his habitually dirty hands and above all, his promiscuity with the dog.

Conservatively one could estimate the cost of treatment of the 72 patients in this series at something greater than £12,000. Eleven new cases were diagnosed in 1957, and 35 patients were domiciled in or adjacent to the metropolis of Melbourne. These facts must surely suggest that more rigid public health control should be exercised in an attempt to eradicate the disease.

Half of the patients in this series had pulmonary hydatid disease without evidence of involvement of other viscera.

TABLE I.

Method of Presentation (72 Cases).

Mode of Onset.	Number of Cases.
Pulmonary:	
Chronic symptoms:	
Chronic or recurrent cough	14
Pain in chest	4 } 19
Dyspnœa	1
Acute symptoms:	
Pneumonia	7
Hæmoptysis	6 } 19
Coughing up of "grape skins"	3
Rupture into pleural cavity	3
Routine X-ray examination	3
Total	41
Abdominal:	
Abdominal pain	15
Abdominal discomfort	1
Abdominal swelling	4
Hepatomegaly as incidental finding	4
Vomiting	2
Rupture into peritoneal cavity	2
Retention of urine	1
Total	29
Cerebral:	
Asthenia	1
Drowsiness	1
Total	2

Considering the series as a whole, 49 of the 72 patients (68%) had pulmonary disease, whereas only 32 (44%) had hydatid cysts in the liver. Five authorities quoted by Sir Harold Dew give the frequency of hepatic cysts as 71% or greater. The high incidence of pulmonary compared to hepatic involvement is therefore very striking. At the Royal Alexandra Hospital for Children, Sydney, 98 patients with hydatid disease have been treated during the past 20 years; 45 of these had pulmonary cysts and 46 had hepatic cysts. Adult series from various other hospitals in Australia show the expected predominance of hepatic cysts. It is suggested, therefore, that in children there is a relative predilection for involvement of the lung. It is tempting to ascribe this to infestation by inhalation, but definite evidence to support this contention is lacking.

TABLE II.

Result of Serological Investigations.

Test Performed and Result.	Number of Cases.
Casoni and complement fixation:	
Positive in both	22
Casoni positive, complement fixation negative	9 } 48
Casoni negative, complement fixation positive	7
Negative in both	10
Casoni:	
Positive	6 } 8
Negative	2
Complement fixation:	
Positive	6 } 7
Negative	1

In considering the symptomatology, attention is drawn to the fact that hæmoptysis occurred in 12 cases; in six of these this symptom was the initial complaint. Hæmoptysis is uncommon in children, and its association with hydatid disease is therefore worthy of comment.

The patient who presented with dyspnœa was a boy, aged 10 years; X-ray films of his chest (Figure I[1]) indicate the fantastic size to which cysts may grow without rupturing or producing other complications. In another case, a diagnosis of pneumonia was made; X-ray examination of the chest revealed an opacity (Figure II) wrongly interpreted as an encysted empyema. Needling resulted in rupture of the cyst (Figure III). The child nearly died, and ultimately required a lobectomy; had thoracotomy been performed instead of thoracocentesis, lobectomy could almost certainly have been avoided. The power of recovery after removal of hydatid cysts is remarkable, and is illustrated by reference to the case of a five-year-old boy with a hydatid cyst of the right lung (Figure IV). The pre-operative X-ray film reveals a large cyst; the bronchogram some years later shows minimal residual bronchiectasis.

The urgency of operation when symptomless hydatid disease is present is also a point worthy of comment. Delay would appear to be unnecessary and in some instances may be dangerous. Figure V illustrates this—it shows the X-ray film of the chest of a nine-year-old girl with hydatid disease of the lung complicated by pyopneumothorax. The presence of lung disease had been observed some months previously, and the parents were advised to seek further attention. This advice was ignored, and the result was an empyema for which decortication of the lung was ultimately required.

The results of serological investigation indicate that in a high percentage of cases a positive response to the Casoni test or the complement fixation test can be expected. Thus in 50 of the 63 cases investigated, this was true. However, each test, separately, produced negative results in one-third of the cases, and both tests produced positive results in less than 50% of the total number. Similar findings have recently been reported by Srouji *et alii* (1958), who reviewed 176 cases at the American University Hospital in Lebanon.

In the majority of cases in this series treatment has followed standard lines, and has proved to be satisfactory. Most of the pulmonary cysts have been treated by removal of the endocyst and internal suture of the ectocyst. The four objects of operation, recently succinctly summarized by Susman (1953) are as follows: (i) complete removal of cyst; (ii) avoidance of soiling and infection; (iii) obliteration of the space which the cyst occupied and closure of bronchial fistulæ; (iv) prompt reexpansion of the lung.

The liver cysts have usually been treated by removal of the endocyst and closure of the ectocyst after it has been filled with saline. The use of formalin has been restricted to the intraabdominal cysts.

Conclusion.

In conclusion, it seems relevant to stress again the cost of treating these 72 patients. Australia shares with South America, Iceland, New Zealand and the Middle East the doubtful distinction of providing relatively large numbers of cases of hydatid disease, and a further plea for stricter measures to prevent perpetuation of this situation would not appear to be out of place. These measures should primarily aim at preventing infestation of the dogs; under such circumstances dogs would not be a menace to humans of any age.

Summary.

1. The clinical features, distribution, presenting symptoms and results of investigations of 72 cases of hydatid disease in children are discussed.

2. In this series boys predominate over girls, and the majority were aged between three and 10 years.

3. The relatively high incidence of pulmonary involvement is contrasted with the situation in adults, in whom hepatic cysts are observed more frequently.

4. Serological investigation is important, but negative results do not exclude the diagnosis.

5. Stricter public health measures are necessary in order to eradicate the disease.

[1] For Figures I-V, see art-paper supplement.

Acknowledgements.

It is a pleasure to record my thanks to the Senior Medical Staff of the Royal Children's Hospital, Melbourne, who generously allowed me to study their cases. Dr. Howard Williams, Director of Clinical Research of the Royal Children's Hospital, was very helpful in criticizing the text of this paper. I am particularly indebted to Mr. Russell Howard, who has taught me a great deal regarding the surgical approach to hydatid disease. Finally, I wish to thank the medical superintendents of the many public hospitals in Australia who provided relevant information.

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Addendum.

Since this review was commenced, a further five patients have been treated; in four, the lung was the site of the disease.

BARON DUPUYTREN.[1]

By J. T. HUESTON, M.S. (Melbourne), F.R.C.S., F.R.A.C.S.,
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> Dupuytren, the man whom, living all admired, few loved and no one understood.
>
> —*Lancet*, 1834.

THE surgical firmament of post-Napoleonic Europe was dominated by two opposing planets, the warm light of Sir Astley Cooper at Guy's Hospital contrasting sharply with the cold brilliance of the Baron Dupuytren at the *Hôtel-Dieu*. It is hard to imagine two men more opposite than the universally popular, charming and compassionate Cooper, and the imposing, cynical, tyrannical and at times terrifying *doyen*, Dupuytren. As some indication of contemporary prestige, however, suffice it to say that *The Lancet*, in his lifetime, named Dupuytren "the most erudite and accomplished surgeon in Europe".

The Hôtel-Dieu.

The hospitals of Paris at the beginning of the nineteenth century comprised 14 hospitals proper and many more *hospices* for infants, the aged and incurables. All were administered through a government-controlled Council General of Hospitals, who made appointments to the staff of a hospital by the method of a *concours*, which involved the competitive presentation of theses by the applicants.

The *Hôtel-Dieu* was the oldest and most celebrated of the Paris hospitals. Situated not far from *Notre-Dame*, it consisted then of two immense buildings, one on each bank of the Seine, connected by a covered wooden bridge. Staffed

[1] Read at a meeting of the Section of Medical History of the Victorian Branch of the British Medical Association, Melbourne, on April 13, 1959.

by three surgeons, 10 physicians, and over 100 non-resident trainees, the hospital in 1833 admitted 16,992 patients, of whom 1783 died—a mortality rate of roughly 10%. It is of interest that an autopsy was performed on every patient who died, which may help to explain the advances in pathology on the continent over the British hospitals where autopsies were less common.

Dupuytren, Breschet and Sanson controlled the five surgical wards, and, of the 250 surgical beds, Dupuytren used about 100 and allowed his juniors the remaining 150. In contrast to the London hospitals, where students had to pay quite high fees for attendance (15 guineas for six months), the clinical lectures at the Paris hospitals were free even to foreign visitors. Dupuytren lectured five times a week to classes of 300 to 400 students, spending at least five hours in the hospital every morning where he met his dressers most punctually at 6 a.m. In his first year as senior surgeon at the *Hôtel-Dieu*, Dupuytren personally treated 846 patients, of whom 368 had major operations, 300 had abscesses opened and 178 had fractures reduced. In the afternoons he conducted his private practice, and it is said that in his later years he saw nearly 10,000 private patients in a year—which is over 30 patients each afternoon.

Dupuytren's Rise.

Guillaume Dupuytren was born on October 5, 1778. The son of a poor advocate in Pierre Buffière, not far from Limoges, he was playing in the village square one day when an officer of a passing cavalry regiment was so impressed by the lad's appearance and bearing that he offered to take him to Paris and arrange for his education at La Manche College. Thus the boy commenced his higher education in 1790 at the age of 12 years; but in 1794, owing to the collapse of the educational institutions of the *ancien régime*, he was obliged to leave college and return the 200 miles to his home on foot.

Returning to Paris the following year, when new medical schools were being established by Fourcroy, Dupuytren was appointed a prosector in anatomy, and from that time his career was one of uninterrupted success. In 1801 he became *chef des travaux anatomiques*; he received his degree in 1803, at the same time becoming assistant surgeon at the *Hôtel-Dieu*. He was made inspector-general of studies of the newly-founded University of France in 1808; but his most brilliant success was in 1812, when he defeated in the *concours* such figures as Roux, Marjolin and Tatra to become professor of operative surgery and successor to the celebrated Sabatier.

It was said by Malgaigne that Dupuytren's thesis on lithotomy, submitted for this *concours*, was regarded for many years as the finest of its kind. He was transferred in 1815 to the chair of clinical surgery, and in 1818 replaced Pelletan as senior surgeon of the *Hôtel-Dieu*. Pelletan's days of office had been numbered since the restoration of the Bourbons in 1815; but his removal was precipitated by his culpable mismanagement of two cases, on which Dupuytren was well able to capitalize. The first case was that of a woman with an extensive malignant growth on the arm, which Dupuytren had forcibly pronounced inoperable. His jealous senior attempted the amputation in a secret operation at which the patient died. The second case involved a Russian officer with a traumatic aneurysm of the external iliac artery, who died when Pelletan incised this mass, having failed to appreciate Dupuytren's advice on proximal ligation in this case. A commission of Russian officers investigated the case and Pelletan was dismissed and replaced by Dupuytren.

It is not irrelevant that Dupuytren's rapid rise from graduation in 1803 to the position of senior surgeon in 1818 took place against the background of the first Empire, and encompassed the whole shattering second era of the Napoleonic campaigns culminating in the Russian *débâcle*, and the subsequent invasion of France and the fall of Paris in 1814 to the European Coalition. Waterloo the following year added again to the stream of casualties returning to the capital where Dupuytren was working. However, the war assisted Dupuytren's progress, not only by providing interesting cases for the development of his skill, but also by eliminating many rivals during the foreign campaigns.

The assassination of the Duc de Berry in 1810 introduced Dupuytren to the Court. He became consulting surgeon to Louis XVIII and was created a Baron, as well as receiving the Order of Knighthood of St. Michael. On the accession of Charles X in 1821, Dupuytren became surgeon-in-chief to the new king, and he held this post until the revolution of 1830.

The immense international repute of this figure was a mixed one, perhaps best summed up in the contemporary epigram:

> First among surgeons,
> *Last among men.*

"First Among Surgeons."

Dupuytren would probably be astounded to find that posterity has associated his name with burns, an ankle fracture and contraction of the palmar fascia. From his writings and his still-very-instructive "*Leçons orales*", it is clear that these three subjects were far from being regarded as his most important. Perhaps, however, it is a tribute to his brilliant teaching style that even such relatively small subjects were presented in so colourful a manner, so clearly and didactically, that they remained etched on the minds of his students and readers. This is the more likely because most other clinical instruction and, indeed, surgical practice involved the stereotyped management of fractures, dislocations and amputations and the opening of abscesses.

Dupuytren regarded an operation as an evil alternative, which nothing short of positive necessity should induce the surgeon to adopt. Like most surgeons of his time, he had tremendous experience and skill in the reduction of fractures and dislocations, and his lecture on the differential diagnosis of the fractured upper end of the humerus from dislocation of the shoulder joint can be warmly recommended to all our modern students of surgery who are being raised on radiology.

Every patient's case history was taken in the same didactic manner with the same three questions:

> *Où souffrez-vous?*
> *Depuis quand souffrez-vous?*
> *D'où vient votre mal?*

The students were thus provided at first hand with a clear, concise history, which was followed by a demonstration of the clinical features of each case and a pronouncement on the patient's care. The astonishing, almost uncanny, power of perception (*le tact médical*) which was attributed to Dupuytren amounted to a very keen clinical sense based on a vast experience—gilded, to be sure, with showmanship of the highest order.

When operation was decided upon, Dupuytren excelled not only in its execution, but in the thoroughness of his preparation of the patient and of his post-operative care, which, he forcibly maintained, determined the success of any procedure. Owing to the lack of general anæsthesia, the extent of the operation was often determined by the preparation of the patient. Hence the frequent suggestion that, if the patient was young and strong, he should be bled daily for up to a week before surgery; this, with the aid of copious draughts of laudanum administered the previous day and strong assistants, enabled major operations to be performed.

From Napoleon's protracted campaigns (in which, it will be recalled, conscripted troops were used for the first time in modern warfare), many thousands of casualties requiring secondary surgery passed through the *Hôtel-Dieu* and other Paris hospitals. Two aspects of this work particularly interested Dupuytren: the definition of the principles of amputation surgery and the surgery of traumatic aneurysm. In a series of brilliant clinical lectures he covered the indications, techniques and complications of amputations at all levels. He was most adamant in teaching conservatism in the common compound fractures and crush injuries, whereas fractures due to gunshot injury he insisted were an indication for

early amputation. ("Here, by deferring operation we lose more lives than we save limbs.") Aneurysms of all sizes came to operation when the conservative ice packs and compression treatment had failed to decrease their size. An advocate of Hunterian proximal ligation, he considered each case carefully and, if necessary, was still prepared to adopt distal ligation, or if proximal compression was possible, even to open the sac and ligate the artery directly. Fascinating descriptions are preserved in his "*Leçons orales*" of intraabdominal ligature of the external iliac

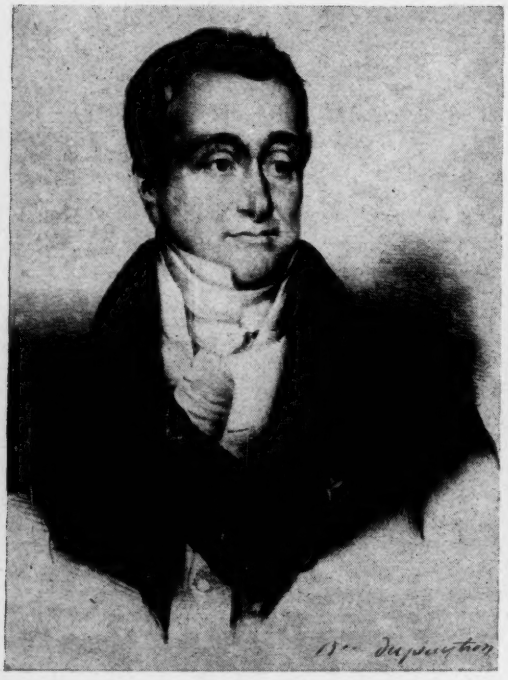

FIGURE I.
Dupuytren.

artery, and of his ligature, after dividing both pectoral muscles, of the third part of the subclavian artery on the first rib. The secret of his success was attributed by Cruveilhier to his sacrificing of brilliance and speed in the interests of safety.

One suspects that if Dupuytren had had the choice of the procedures for which he should be remembered, he would have nominated his cure for lachrymal fistula, his operation for amputation of the mandible, and his cure of the artificial anus. Untreated pyogenic or venereal infection of the lachrymal sac led sooner or later to naso-lachrymal duct stenosis, lachrymal abscess and, finally, an external lachrymal fistula from the sac. In a spectacular three-minute operation (with the patient strapped into a chair facing a well-lit window and the head steadied on the assistant's chest) the sac was opened with a single stroke, and in a second movement a special curved gold or silver cannula was thrust, on a trochar, through the medial wall of the sac into the nasal cavity, where it provided drainage and created a permanent internal fistula, thus allowing the external fistula to close. Although a cruder version of this procedure had been described half a century earlier by Pott, and in fact had been performed with the cautery by Cheselden, it is clear that Dupuytren was proud of his improvements in technique, and he claimed to have performed the operation 3000 times, obtaining a cure of the fistula in 19 of every 20 patients. As we would expect, the metal cannula frequently required removal later, and this disadvantage of the operation prevented its being accepted in England.

Dupuytren's technique in resection of the mandible for malignant disease was swiftly to split the lower lip in the mid-line to the hyoid with a single stroke, reflect the two lateral flaps close to the mandible in order to avoid the inferior labial arteries and, after sawing through both sides, free the mandible from the floor of the mouth with a sweep of the straight bistoury. He outlined the main complications and how they should be anticipated; namely, asphyxia due to falling back of the tongue, secondary hæmorrhage, cellulitis, and œdema of the glottis.

The so-called "artificial anus" (*anus contre nature*) was an external fæcal fistula due to sloughing of a strangulated hernia, and for this hitherto incurable affliction Dupuytren

Episode for a Guy's Pageant.

"Sir Astley's fame was European, so that distinguished foreign surgeons never failed to visit him at the Hospital. We read of Dupuytren going round the wards with him. . . . . When he took leave he saluted the worthy baronet on each cheek. The manner in which Sir Astley submitted to the ceremony afforded no small amusement to the pupils standing round."—WILKS AND BETTANY'S HISTORY.

FIGURE II.

Dupuytren embracing Sir Astley Cooper on the occasion of his visit to Guy's Hospital in 1832.

The English baronet was somewhat taken aback but countered with, "Well, never mind, I'm even with you, for when I was in Paris I kissed your daughter."

(*Lancet* (1829) 2: 224.)

(Cartoon reproduced by kind permission of *Guy's Hospital Gazette.*)

designed an enterotome which was the prototype of the colostomy crushing-clamp still used today. Surgeons would visit his clinic from Britain, Europe and America, often specifically to witness a demonstration of the insertion of this enterotome. Many glowing accounts are found in the contemporary medical literature of successful cures effected by Dupuytren's masterly use of this clamp.

Nearly every subject in surgery has been covered in Dupuytren's recorded lectures, from ingrowing toenails to cerebral abscess, from foreign bodies in the trachea to cysts on the mandible, and from ranula to the advantages of conservative treatment of abscesses occurring in the right iliac fossa. Although he did not serve in Napoleon's invading armies, he was not without experience in the

primary care of war wounds. On March 3, 1814, during the fall of Paris to the European Coalition, it is recorded that Dupuytren crossed the city with his internes from the *Hôtel-Dieu* to care for the wounded beside the barricades, under heavy fire from the enemy. Again in the revolution of 1830, he was constantly at the *Hôtel-Dieu*, with such splendid devotion to his work that a public proclamation was made to honour it.

The retraction of the palmar fascia by the description of which Dupuytren has been immortalized was, by a curious twist of fate, one of the aspects of his work which was not as original as he claimed, and for which a better operation than his had been described some years earlier. His description of the clinical picture which immortalized the *marchand du vin en gros* and his *cocher d'environ quarante ans* was so accurate, and so beautifully demonstrated by his dissection of the amputated hand that the condition was due to contraction of the palmar aponeurosis, that it is small wonder that his name will be permanently associated with this condition. However, his rival Sir Astley Cooper (whom he quoted only as having told one of his patients that nothing could be done for his hand) had a decade earlier, in 1822, described this contracture, attributed it correctly to the palmar aponeurosis and recommended subcutaneous fasciotomy for its cure. This latter procedure now appeals more than the multiple incisions through both skin and fascia used by Dupuytren, which he followed with prolonged splintage in the straightened position, but which we should now expect to allow an early recurrence of the deformity.

"Last Among Men."

Sheer ability alone would not have placed Dupuytren in his preeminent position at the *Hôtel-Dieu* by the age of 41 years. He possessed, along with his surgical skill, an immense capacity for self-advancement or, more concisely, *savoir vivre*, which could not help but arouse the envy and antagonism of his contemporaries and competitors.

Had he been great enough to ignore the malice of the envious, there might have been less reason for the stinging epigram just quoted. Whether because of his desertion by his wife, or because of an innate paranoid tendency, Dupuytren conveyed, in all his dealings with others, an impression of cynicism and universal distrust. Mediocrity was his greatest fear in life, and his favourite maxim—*"ce qu'il faut craindre avant tout c'est d'être un homme médiocre"* ("above all things avoid being an insignificant person")—suggests a personality always striving to impress with its superiority, if necessary at the expense of humiliating others or twisting facts to fit its fancy.

An imperious attitude towards students, dressers and patients alike characterized his clinics: he was "cold, capricious and tyrannical to the last degree". Perhaps the most disturbing story of this brutal side of the man was his treatment of the mother of a child whose leg he was about to amputate. Forcing her way into the room, the distraught mother interrupted the operation, whereupon Dupuytren completely lost control of himself and literally kicked the woman out of the room with a *"coup de pied dans le derrière"*.

The currently fashionable cult of physiognomy is evident in this contemporary description of Dupuytren's face.

> Viewing the upper part of his face, and particularly his broad fair forehead covered by a thin white chevelure, the figure was that of a man imbued with feelings of benevolence and accustomed to exert the most untiring patience. But soon would the impression be destroyed by a sudden curl of the lip, an almost inperceptible compression of the mouth, a fastidious shrug of the shoulders—tokens of the mental storm within, which he was disdainful to show.

Eccentric, but distinctive, in his dress, Dupuytren at all times wore a little round-cut green body coat and, in the hospital, a small green cloth cap "of a cut altogether original" with its brim turned up from his forehead. His impressive entry to the lecture theatre with a slow, "almost jesuistic" step, right hand thrust into the bosom of his coat, must have been offset a little by his habit of constantly gnawing at the fingernails of his left hand, which hardly left his mouth except to operate.

"The Brigand of the *Hôtel-Dieu*", as he was dubbed by Lisfranc, brooked no rival near the throne and lost no opportunity to scorn the work of others. His *"Leçons orales"* often furnish such a passage as the following: "Those who have seen this patient can appreciate the immense service conferred on such a one whom so many practitioners had abandoned." Virgil classed surgery among the dumb arts, when he said of Japes *"maluit et mutas agitare inglorius artes"*, but it was maliciously said of Dupuytren by a contemporary writer that he proved the Roman poet wrong by showing that surgery could furnish a strong voice for self-advertisement.

The immense fortune amassed from his large private practice was attributable both to his court connexions and allegedly also to his determined method of fee collection. Dupuytren had a code of signals by which he notified his hall-porter if a client had failed to pay cash, and it is related that an English lord who had forgotten to bring his wallet was apprehended by the porter on leaving and made to leave his gold watch as security.

The great Dominican preacher, Lacordaire, in a sermon at *Notre-Dame* in 1851, told of a more compassionate Dupuytren.

> A poor country priest suffering from a fungating tumour of the neck was sent to him as a last resort by his parishioners, who had made up a little purse among them for the purpose. The great surgeon handled the mass and probed its base, with his usual disregard for the pain he was causing, and finally announced; "There is no hope for you; you must die." The priest thanked him, and expressed his resignation to the will of God with such gentle fortitude that the surgeon was touched to compassion and decided to operate in the faint hope of success. This hope was granted, the priest survived, and on hearing many years later of Dupuytren's illness he set out for Paris on foot, to arrive in time to administer the last consolations of religion to the dying man.

Final Illness.

Dupuytren had planned to give up surgery at the age of 60 years, but fate overtook him two years before he could retire. On November 5, 1833, a mild cerebral thrombosis produced a right-sided facial palsy. Cruveilhier's description of the onset of this palsy during a morning clinic is impressive: "With the air escaping from his mouth thus upsetting his speech he supported his cheek with his hand to finish the lession." Determined as he was to carry on his work, it was only by the greatest persuasion that Dupuytren's daughter was able to take him to Naples, where he rested for the winter. Six months later he resumed his commitments in Paris amid "warm plaudits" from his pupils, whose instruction he could take now only twice weekly while Sanson continued on the other days. However, he showed but a glimmer of his former brilliance. Hampered by some residual speech defect and fatigued beyond his waning physical powers, he struggled through a few months until his death from pneumonia and empyema occurred at 3 a.m. on February 8, 1835. He lived for 58 years.

His courage and sang-froid never deserted him, although he knew death was imminent. The evening before his death he insisted on having the paper read to him as usual so that he might carry the most recent news out of this world (*"Afin de porter là-haut des nouvelles de ce monde"*).

As was the custom, Dupuytren bequeathed his body for examination, which was performed by two of his old pupils, Broussais and Cruveilhier; their lengthy report confirmed the existence of lung and cerebral changes, although the brain was a little dried out, having first been given to an artist to prepare a cast. In his will he left 200,000 francs, which were used in the founding of the *Musée Dupuytren*. The remainder of his fortune, amounting to 7,000,000 francs (£280,000 sterling), he left to his only daughter.

At Dupuytren's funeral on February 10, the funeral car was drawn by his students from the Church of St.

Eustache to the tomb in *Père-Lachaise*, where orations were delivered by Orfila, Larrey, Bouillard, Royer-Collard and his interne at the *Hôtel-Dieu*, Tessier. No record is available of these addresses, but a curious note is struck by his obituary in *The Lancet* which must convey some of the disturbing atmosphere at his funeral.

> The void which he has left is immense. Who can fill it? Who can succeed to that chair with éclat, which has been filled by Dessault, Pelletan and Dupuytren? That it will even be filled to the best advantage which circumstances admit, we have reason to disbelieve. Intrigue is at work, and there is cause to engender a fear amongst the profession in Paris, that the clinical instruction in the Hôtel-Dieu will be entrusted to one of the worst clinical lecturers in the capital.

Appendix: Published Works of Dupuytren.

"Propositions sur quelque points d'anatomie, de physiologie, et d'anatomie pathologique." 32 pp. Paris, an XII, 1803, v. 39.

"Expérience touchant l'influence que les nerfs du poumon exercent sur la respiration'. 24 pp. Paris, 1807. (Repr. from: Biblioth, med., Par., 1807, XVII, 3-24.)

"Lithotomie". 55 pp. Paris, Lebegne, 1812. (Also in: Paris theses, v. 94, No. 185 Concours.)

"Projet pour l'organization de l'art de guérir". 35 pp. Paris, 1816.

"Considérations générales sur l'épidémie qui ravagea Barcélone en 1821." Paris, F. Didot, 1826.

"Leçons sur les étranglements des hernies par le collet du sac herniaire." 51 pp. Paris, J-B. Baillière, 1832. (Repr. from: J. Univ. et hebd. de med. et chir. prat., Par., 1832, IX.)

"Lettre et leçon sur le siège, la nature et la traitement du choléra morbus." (Collected and published by A. Paillard and Marx.) 31 pp. Paris, J-B. Baillière, 1832.

"Leçons orales de clinique chirurgicale, faites a l'Hôtel-Dieu de Paris." 4 vols. Paris, Germer-Ballière, 1832-4. (Revised by Brierre de Boismont and Marx, 6 vols. 1839.)

"Traite théorique et pratique des blessures par armes de guerre." (Published under the direction of A. Paillard and Marx.) 2 vols. 522 and 527 pp. Paris, J-B. Baillière, 1834.

"Mémoire sur une manière nouvelle de pratiquer l'opération de la pierre.' (Finished and published by J-L. Sanson and L-J. Begin.) 50 pp. Paris, J-B. Baillière, 1836.

EPILEPSY IN THE MENTAL HOSPITAL.

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AND

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THE management of the epileptic patient in the mental hospital has always been a problem. It is, in fact, a problem of total management, for the patient is not admitted to the mental hospital just because he has fits. Often there are associated personality defects or psychotic episodes (automatisms, psychic equivalents, psychomotor epilepsy, temporal lobe epilepsy, dreamy states), sometimes the epilepsy is merely an additional handicap to an intellectually backward person, or the fits may be a symptom of some underlying organic cerebral disease process.

Over a three-year period we have made a study of epileptics. In this paper we wish to report the results of some drug trials, made as part of this study. Because the epileptic patient in the mental hospital presents a different problem from the epileptic in the community, we do not believe that our results will necessarily have the same application outside the mental hospital environment.

To emphasize this difference, the patients in this study must be defined. There has been in the past an administrative policy to transfer female epileptic patients to this hospital, so that out of 600 patients there have been 105 to 115 epileptics. There have been 35 to 45 male epileptics in a population of 340 men. These patients can readily be classified into three groups following Liddell (1953), though there is also a small group of schizophrenic patients who secondarily develop epileptic fits. Other organic psychoses with symptomatic epilepsy can be grouped in Liddell's second category with dementia.

The accompanying Tables I and II are given so that a clearer picture may be obtained of the nature of problem of epilepsy in the hospital. Age and duration of illness are tabulated, together with comparable figures from Liddell's paper.

TABLE I.

Subjects.	Mental Defect.	Organic Process.	Schizophrenia.	Automatisms.	Total.
Liddell's patients:					
Male	9 } 18	3 } 11	—	13 } 18	47
Female ..	9 }	8 }		5 }	
Ballarat patients:					
Male	10 } 36	11 } 17	7 } 17	16 } 80	150
Female ..	26 }	6 }	10 }	64 }	

The transferring of epileptics to this hospital has decreased in the last few years on account of overcrowding. As a result, there is a residual population of long-standing epileptics. It is this population which has served as the basis for the three-year study here reported.

In order to describe the results adequately, it is necessary to outline in more detail the arrangement of epileptics in the hospital. Sixty female patients are nursed in one large ward of 120 beds (Ward 60) and 25 in another similar ward (Ward 25), and the remaining 25 are equally distributed in geriatric, chronic long-stay and infirmary wards. Of the epileptics in Ward 60, about a quarter have mental defect, but in Ward 25 there are only one or two. The precise figures for ward and total numbers and distribution have varied to a small degree during the three years. In the male division, the epileptic patients are fairly evenly distributed through six wards and classified entirely on their merits amongst other psychotic patients.

TABLE II.

Condition.	Subjects.	Mean Age on Admission. (Years.)	Mean Stay in Hospital. (Years.)	Mean Age in 1957. (Years.)	Mean Age, Liddell's Series. (Years.)
Automatisms	Males.	29·8	13·7	43·5	38·75
	Females.	33·5	16·25	48·75	53·4
Organic process	Males.	44·8	9·9	54·7	54·3
	Females.	56	9·0	65	65·8
Schizophrenia	Males.	38	19	57	—
	Females.	27	19	46	—
Mental defect	Males.	21·1	26·3	47·4	38·2
	Females	24·4	17·1	41·5	50·6

While most of the drug trials have been carried out in one ward only, one was carried out in a random manner throughout the female division. The various trials to be described were carried out on patients with automatisms to the exclusion of others. However, when the results were compared with the rest of the epileptics in the ward, no such exclusion was made.

Primidone.

The first drug trial was with primidone, and was carried out on 10 patients from Ward 60, all of whom suffered from "temporal lobe epilepsy" with regard to personality and clinical manifestations. The mean duration of the trial was eleven months (standard deviation ± 6·4 months);

but in three cases interruption was necessary because of increasing drowsiness and ataxia four, five and seven months after a dose of 1·5 grammes per day had been reached. In this respect our observations agree closely with those of Wilson and Hodgson (1954), who also noted the late development of ataxia and slurred speech after a period of normal adjustment.

All those who showed overt aggressiveness before primidone continued to do so whilst receiving primidone. None became overtly aggressive who had not been so before. The mean monthly number of sezures for this group for the preceding 12 months was 4·57, for the period of primidone therapy 4·22, and for the 12 months after the cessation of the drug 3·54. The figures for the rest of Ward 60 as a whole for the same period were 1·23, 1·25 and 0·81 seizures per patient per month. Whilst the figures before and after primidone therapy approach statistical significance at the 5% level, the differences with the figures for the actual trial are not noteworthy. It seems rather that both the figures for primidone and the ward as a whole reflect some general tendency, and do not contribute to any reliable study of the effect of primidone. Some suggestions about this will be made later.

"Phenurone."

The observations with "Phenurone" were carried out in random manner on nine patients selected from various wards in the female division, but without any suggestion that they constituted a special group as with the other drugs used. The usual records of behaviour and fits were kept, and the nurses were instructed in the danger signs in view of the known toxicity of "Phenurone".

The patients were selected on the basis of personality deviation, with the emphasis on aggressiveness and interpersonal difficulties; in three the actual seizures were well controlled. For the first month after starting "Phenurone" therapy on various dates over a period of two months, only two patients recorded any fits. Thereafter the tendency to have fits returned, and four months later only three were still taking the drug and receiving continued benefit from it.

The improvement in personality was equally dramatic and equally evanescent. Some formerly persistently quarrelsome and argumentative patients were models of good behaviour and cooperation with everyone, whilst others showed little change in their behaviour. One patient showed a gratifying response to the drug, which was not sustained, again responded to an increased dose only to relapse, and again responded to a further increase but only temporarily. Finally she was changed back to her former medication without any further deterioration.

The actual figures for the mean monthly seizure frequency for the preceding six months (except for two patients recently returned from a period of trial) was 2·28, and for the period of trial (mean duration four months) the figure was 0·72; for the six cases in which "Phenurone" therapy was discontinued, the mean frequency over the next six months was 1·15 when they were placed back on their former régime or with some modification of the basic phenobarbitone, "Phemitone", phenytoin or "Tridione". The figures before and during "Phenurone" therapy are statistically significant at the 5% level; but this may be due to the almost complete absence of fits for the first month, rather than to any real sustained benefit. The before and after figures only reflect the general tendency in the female division, because the mean monthly seizure frequencies for approximately the same periods were 1·22, 1·32 and 0·79.

Reserpine.

Two groups of patients were given a trial with reserpine and chlorpromazine. Both these studies were part of a departmental study with these drugs, and some conclusions will be quoted from this source (Stoller, 1958) together with some further observations more relevant to this paper.

The period of observation was 90 days. Reserpine was given, the dosage starting at 9 mg. (4 mg. by mouth and 5 mg. by intramuscular injection) and being increased by 5 mg. intramuscularly after three weeks to 14 mg. and then at weekly intervals to a maximum of 29 mg. per day. Of the 10 patients, only four continued to the level of 29 mg.; others were stabilized at lower doses. After 29 mg. had been reached, the dosage was reduced to an optimum level or discontinued after 90 days when no benefit had accrued. Other anti-epileptic medication was continued throughout.

Some initial improvement was noted in the behaviour of half the patients whilst they were taking the lower doses, particularly with respect to their nursing management. Side-effects became more apparent on doses of 14 mg. and above. Depression appeared in four patients, one of whom made an attempt at suicide. There was a loss of appetite at some stage in nine of the patients, reflected in the fact that seven lost weight (average loss 9·5 lb. range 2 to 15 lb.).

Eight became somewhat drowsy on the higher doses, and less able to carry on their usual routines and occupations, preferring to laze about and doze all day. Two became over-active, one with a great deal of psychomotor agitation necessitating a short period of seclusion. Reduction of dose did not improve matters, and the drug was withdrawn on the fifty-sixth day. Another patient complained that "her legs were walking her". She could not rest, and spent much time walking in circles. This side effect disappeared when the dose was reduced.

Only mild evidence of Parkinsonism was noted, and no tremor developed. Facial expression was noticeably reduced in five cases. This was associated with an obvious degree of facial œdema, most marked in the periorbital tissues.

Three patients were considered to have shown useful improvement at the end of the period; two had carried out the full régime and the dosage was stabilized at 16 mg., but the third had developed side effects at 14 mg. and the dosage had been reduced to 4 mg. per day. In view of the untoward effect in the majority of cases, the general impression with this study of reserpine was one of dissatisfaction. However, the actual record of fits shows an improvement. The reserpine group showed a mean monthly seizure frequency for the preceding 12 months of 1·34 fits per person. For the three months of the trial it was 0·73. The figures for the remaining epileptics in the same ward for the same periods were 1·28 and 0·98. For the next nine months the drug group had a mean monthly frequency of 1·29, the rest of the ward 0·98.

Chlorpromazine.

A very similar plan was used with the chlorpromazine trials, which were carried out on 14 epileptics in Ward 60. Only 10 completed the study and these 10 are the basis for the observations. In four of these cases the trial was terminated because of Parkinsonism, ataxia and intercurrent infections.

The dosage in this study started at 300 mg. per day for three weeks, and then increased by 300 mg. per week to a maximum of 1200 mg. and decreased again by 300 mg. per week; the trial was discontinued at the end of 90 days. Some patients were later stabilized on the optimum dose. It was established that some real and positive change did occur during the treatment period; the most disturbed gained the most, but still tended to maintain their relative ranking in the group as regards disturbance. There was little further gain after the initial period on the low dose of 300 mg. The improvement noted was apparent in all rated variables, grouped under the titles of personal adjustment, manageability and social adjustment (Stoller). There is, in the absence of the usual control groups, doubt as to the genesis of this improvement.

During this trial, the incidence of recorded fits showed quite remarkable changes. For the previous 12 months

TABLE III.

Drug.	Where Used.	Number in Group.	Mean Monthly Seizure Frequency.[1]					
			For 12 Months Before Test.		During Test (3 to 12 Months).		After Test (6 to 12 Months).	
			A.	B.	A.	B.	A.	B.
Primidone	Ward 60.	10	4·57	1·23	4·22	1·25	3·54	0·81
"Phenurone" ..	All female wards (105).	9	2·28[2]	1·22	0·72[2]	1·32	1·15	0·79
Chlorpromazine ..	Ward 60.	10	1·42	0·99	0·51	0·40	1·20	0·74
Reserpine	Ward 25.	10	1·34	1·28	0·73	0·99	1·29	0·98

[1] A, test groups; B, remainder of ward(s).

[2] Significantly different at the 5% level. See text for exact duration of periods of observation.

the mean monthly seizure frequency for this group of 10 patients was 1·42, for the whole ward 1·06, and for the rest of the epileptic patients in the ward 0·99. For the 90-day test period the group on the drug averaged 0·51 fit per patient per month, but the figure for the remainder of the epileptics improved to 0·40. During the next month, in which the chlorpromazine was entirely suspended for two to three weeks, the figures were 0·33 and 0·27. Finally, for the next six months the drug group averaged 1·20 and the non-test epileptics 0·74. (These figures are calculated for nine patients—one died during the last six-month period and has been excluded from these figures.)

Review of Changes.

The drug trials described in the preceding sections were in the case of the "tranquillizers" superimposed upon an established régime of anticonvulsive therapy. "Phenurone" was prescribed on a similar basis. Primidone was substituted for phenobarbitone, but phenytoin, if already prescribed, was continued throughout the primidone trial. A combination of phenytoin and phenobarbitone was the usual régime used, with "Phemitone" substituted for phenobarbitone when the latter was deemed to be less satisfactory. A review of the records for the years prior to this study indicated that at times "Phemitone" substitution for phenobarbitone or vice versa resulted in marked reduction in the frequency of fits.

The data regarding the actual numbers of recorded fits have been brought together in Table III. In all instances the result is an improvement in the figures after the test for both patients given the drugs and others in the various wards. There would seem to be several possible explanations for this. On one hand, there has been evident a general tendency to reduction in fits throughout the three years, which is reflected in the studies here reported. The general attention and individual care in the wards may have increased as a result of the observations, and these factors could have had a beneficial result. This implies that the actual fit has some relationship to the interpersonal relationships between patients and staff.

In this respect the chlorpromazine trial is of particular interest, since the over-all reduction in seizure frequency during and immediately after the trial period was considerable. This reduction may be correlated with the general observation that the more disturbed patients become less "turbulent" while receiving chlorpromazine. This resulted in a lowering of the interpersonal tension of the ward as a whole. Thus, although it is not possible to draw any conclusions regarding the effect of chlorpromazine on the individual epileptic, it may be suggested that the drug has some beneficial though indirect effect on the epileptic group as a whole. The interpersonal tensions in the ward may be "tranquillized", with a lowered fit frequency as a consequence. A similar conclusion was suggested by Andermann and Lindsay (1955) in a study of chlorpromazine with chronic "turbulent" schizophrenic patients.

It seems difficult to give a satisfactory explanation for these figures in terms of a disease reacting specifically to a given remedy. The actual fit is, of course, not the disease, but nevertheless a measurable symptom, in which reduction has occurred both in those given the drug and in those not so treated. The effect is not confined to the group in which the drug is administered.

During the three years in the hospital, many of the accepted methods of social psychiatry have been applied with patient-run clubs and activties, all tending towards greater freedom and responsibility. This, as Bell (1955) has noted, does lead to benefit in epileptic patients. If this was a satisfactory explanation, it nevertheless does involve a consideration of interpersonal relationships, for there are increasing opportunities for patient activities, and a changing attitude on the part of the staff.

As a result of these considerations, we have made some investigations into staff and patient attitudes to epilepsy and epileptics, to be reported elsewhere. These raise many questions rather than providing any simple solution. It seems essential that the epileptic patient in the hospital be viewed as subject to and reacting to the totality of his "here and now" environment.

Summary.

A report is made of the result from four drug trials in mental hospital epileptic patients, using "Primidone", "Phenurone", reserpine and chlorpromazine. The over-all improvement with all the drugs and in all control groups has led us to examine other ways in which the amelioration could have been produced. Some of these are briefly discussed.

Acknowledgements.

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Reviews.

Strong and Elwyn's Human Neuroanatomy. By R. C. Truex; fourth edition; 1959. Baltimore: The Williams and Wilkins Company. 10" × 6¾", pp. 524, with 363 illustrations. Price: £5 10s.

THE fourth edition of this well-known book has been revised by Dr. Truex, who is Professor of Anatomy at the Hahnemann Medical College. Previous reviews have noted the work as sound and well illustrated, even if not

particularly outstanding among a large number of books on this subject. In his revision Dr. Truex has made a number of changes. Now, after an introduction to comparative neurology, histology and embryology, the student is presented—as he logically should be—with an account of the gross anatomy, meninges and blood supply of the central nervous system before plunging into the intricacies of internal organization. This preliminary orientation is valuable, although the section on blood supply is sketchy and inadequate. The remainder of the text follows much the usual order; but some parts have been rewritten and shortened and a number of new illustrations are included. A change of policy here has distributed the atlas of Weigert sections of former editions throughout the text in this edition. No doubt the author feels that this makes the book more generally useful; nevertheless, there is a good deal to be said for having an atlas as such for quick comparison of levels. We sympathize with his confession of inability to include all the latest contributions to neurology, but consider that on the whole he has coped reasonably well—at least for an undergraduate text. The book is well produced and illustrated, and it remains a sound work of its kind.

Somatic Complications Following Legal Abortion. By J. Lindahl; 1959. London: William Heinemann Medical Books Limited. 9¼" × 6¼", pp. 182. Price: 42s.

THIS book is a report of an investigation designed to elucidate the incidence and character of somatic disturbances which followed legal abortion in Sweden.

The investigation consisted of examining the case records of 1188 patients who had undergone legal abortions and noting the immediate complications following the operation. At least a year after the operation the author examined 1013 of these women.

The abortion was performed in 989 cases by vaginal hysterotomy, in 51 by dilatation of the cervix and curettage, and in 130 by abdominal hysterotomy and ligation of the Fallopian tubes. There was only one death in the series, and relatively serious immediate complications occured in 3·6% of the total series.

The positive findings in the follow-up examination were as follows. Endometriosis in the vaginal scar occurred in 15% of cases. Deformation of the isthmus with a few fine fistulæ between the uterine cavity and the anterior fornix occurred in 5% of cases. There was no evidence of incompetent cervix, and the spontaneous abortion rate was not raised when subsequent pregnancies occurred, of which there were 265. There was a very low incidence of salpingitis following the operation, and the incidence of impaired tubal function was less than 1·6%.

The author states his findings and leaves the reader to draw his own conclusions as to the magnitude of somatic complications following legal abortion. As vaginal hysterotomy as a method of abortion is rarely used in Australia, the end results are rather academic.

A Handbook for Dissectors. By J. C. Boileau Grant; fifth edition; 1959. Baltimore: The Williams and Wilkins Company. 8" × 5¼", pp. 456, with illustrations. Price: $5.00.

THIS volume of dissecting-room instructions is written for use with "A Method of Anatomy", and is by the same author (with the help of Dr. Freda Richardson who revised the chapter on brain dissection). The new Paris nomenclature (N.A.P.) is used, common or important older terms being included as well; measurements are given in inches, although fingers'-breadths still appear here and there.

In this edition there have been notable improvements, and the author has produced an eminently satisfactory volume of succinct instructions for the dissection of the human body.

More descriptive detail has been added to the text, and this, coupled with the continued use of heavy type to emphasize important structures, will make the student's task easier. More important is the addition of over 100 figures; dissectors, especially in their earlier days, tend to make frequent use of diagrams to find their way about, and although the majority of the new figures are borrowed from the author's textbook, their ready availability with the dissecting instructions will be welcome. Some diagrams would be more useful with complete labelling of structures. The chapter on brain dissection suffers from lack of illustrations.

The loose-leaf binding has the distinct advantage that the book will lie flat on the table when opened at any page; but the method of securing individual pages does not offer a very long life under ordinary dissecting-room conditions.

The Cranial Nerves: Anatomy and Anatomico-Clinical Correlations. By Alf Brodal, M.D.; 1959. Oxford: Blackwell Scientific Publications. 7¼" × 4½", pp. 144, with 25 illustrations. Price: 15s. (Abroad).

PROFESSOR BRODAL is well known as an experimental neuroanatomist with a leaning to clinical applications of his work. The production of a work such as this from his pen does not, therefore, come as a surprise. After a preliminary account of the nerves in general, the cranial nerves are taken up in order, starting with the twelfth. Each is discussed, with a brief account of its origin, course, distribution and functional make-up, illustrated by a simple line diagram; an account of the effects of damage follows. All this is managed very concisely and clearly; but some of the diagrams contain a little too much detail for the size of reproduction, while some others are perhaps over-simplified. The volume concludes with a brief account of some common neurological syndromes. There are a limited list of references and an index. Taken all round, this book should be a great help to students in their clinical neurology, and to practitioners looking for an easy, quick key to the cranial nerves. It may well be said that anybody who knows the book will tackle lesions of the cranial nerves with some confidence. On the other hand, the practitioner has to work back from signs and symptoms to the lesion, and a short section on differential diagnosis would have considerably enhanced the value of the work. Despite that, this modest little book will undoubtedly prove of great assistance in the field it covers, and can be recommended with confidence.

Sigmund Freud's Mission: An Analysis of His Personality and Influence. By E. Fromm; 1959. London: George Allen & Unwin. 7¼" × 4¾", pp. 127. Price: 12s. 6d.

IN "Sigmund Freud's Mission", Erich Fromm essays an analysis of Freud's motives. Fromm suggests that increasing social and economic instability in the Austrian Empire doomed to disintegration was a factor in developing Freud's self-reliance and trust in reason. Yet this paradoxical personality had a morbid fear of poverty and would never travel alone. He dominated his family early in his childhood, and his dislike of music led to the termination of his brothers' and sisters' musical education. Freud's attitude towards his parents was one of dependence on his mother and rebellion against his father. Fromm suggests that had his father been a greater man Freud might have submitted to him. Freud treated his wife as a typical continental *Hausfrau*, had little interest in women individually, and had no use for their emancipation.

His attitude towards co-workers and assistants was one of dependence until his autocratic behaviour led to their secession. From a method of psychiatric investigation and treatment, Freud evolved the idea of a movement to spread his theories in the fields of sociology and philosophy. But the International Fraternity for Ethics and Culture gave place to the International Psychoanalytic Association. Freud had hoped that his work on the unconscious would help mankind to control its irrational passions by reason. Two World Wars and a considerable extension of sexual licence threatened to shatter such aspirations. Yet, in spite of economic loss, political persecution and an agonizing illness in his last years, Freud courageously maintained his ideals. While not belittling Freud's achievements, Dr. Fromm presents his subject as a supreme egotist whose theories were inspired by his neuroticism.

Smoking, the Cancer Controversy: Some Attempts to Assess the Evidence. By Sir Ronald A. Fisher, Sc.D., F.R.S.; 1959. Edinburgh and London: Oliver and Boyd. Adelaide: Rigby Limited. 8½" × 5½", pp. 48. Price: 2s. 6d.

SIR RONALD FISHER believes that smoking is not the cause of the recent increases in the lung cancer death rates. He is much impressed by evidence that smoking habits are genetically determined. He concedes that lung cancer deaths are associated with heavy smoking, but believes that they are both dependent on some other, more fundamental, cause. Doll and Hill found that, for a fixed consumption of cigarettes per day, lung cancer patients "inhaled" less than the controls. This might have been thought crucial; but Doll and Hill, whose work is not documented here, have found it prudent to drop this question from later surveys. Fisher criticizes this at great length. (We may remark that few people know

whether they are inhalers or not.) Pages 26 to 38 seem to us to be irrelevant.

Sir Ronald Fisher has preferred to discredit a minor part of the work done on lung cancer rather than to survey the whole problem. We can only hope that further epidemiological work will produce an answer to this important question.

The Physiology and Treatment of Peptic Ulcer. Edited by J. Garrott Allen, et alii; 1959. Illinois: The University of Chicago Press. 9¼" × 6", pp. 250, with many illustrations. Price: $7.50.

THIS well produced volume is in the form of a "Festschrift" in honour of Lester Dragtedt, by 12 of his colleagues and former students from the University of Chicago, at the time of his retirement from its chair of surgery. The contents present a summary of the experimental and clinical approach to peptic ulcer by the Chicago School, which in the past twenty years has produced significant advances in both fields. The book does not purport to be a review of all aspects of the disease, and some of the views, especially those on the role of the vagus nerve in the genesis of duodenal ulcer, do not now receive full general acceptance. Dragstedt's view of the importance of gastrin in the production of gastric ulcer follows an excellent review by Woodward on the physiology of the gastric phase of gastric secretion integrating the available experimental evidence. Divergent views on the place of surgery in gastric ulcer appearing in succeeding chapters perpetuate the unfortunate disparity between medical and surgical thinking towards the disease. Multiple authorship and overlap of subject matter are apparent particularly in the sections dealing with the cephalic phase of gastric secretion and the rationale and methods of treatment of duodenal ulcer. Whereas Kirsner gives an excellently summarized approach to the medical aspect, the present unsatisfactory position on surgical management is delineated rather than answered. An innovation in a book of this type is the chapter by Hodges on radiological methods in diagnosis and treatment. The limited appeal of the accent in subject matter and approach is offset by the excellent documentation of the material presented, and the book will probably stand as a record of two decades of intensive work and thought at this leading centre. Although some chapters can be read with profit, the volume does not clamour for purchase.

Hypnosis: Fact and Fiction. By F. L. Marcuse; 1959. Mitcham, Victoria: Penguin Books Pty. Ltd. 7" × 4", pp. 224. Price: 5s. 6d.

DR. F. L. MARCUSE, Professor of Psychology at Washington State University, Pullman, U.S.A., is well known for his work in hypnosis. The present volume in the "Pelican" series is intended primarily for the lay reader; but it contains much that will be of interest both for students, and for those who are professionally concerned with aspects of clinical or experimental hypnosis.

The author states that the aim of the book is simply to separate fiction from fact in hypnosis. This, of course, is no new theme. Braid, in his book "Neurypnology" (1843) quotes the motto: "Unlimited scepticism is equally the child of imbecility as implicit credulity." Over the years, many authors have sought to sift the grain from the straw in the field of hypnosis. It was a favourite subject of Elliotson in his papers in *The Zoist*; and so it has continued until the present day. Dr. Marcuse has brought these matters up to date, and presents a simple account for the layman of what is known and what is not known. Even more important than this, he indicates those areas where the evidence still appears to be conflicting.

As is proper in a book of this nature, there is no discussion of either the more complicated theoretical aspects of hypnosis or the clinical technique of hypnotherapy. It is not intended as a guide for the physician who would use hypnosis in his practice, but rather as a source of information for those who read for the general interest in the subject.

In sifting fact and fiction, the author rightly pays a good deal of attention to a discussion of the dangers of hypnosis. He dispels the common misconceptions of ignorance, and warns of the dangers of unfulfilled post-hypnotic suggestion. But it would seem that he rather neglects the important dangers of perverse motivation and of spontaneous abreaction with sudden awareness of unconscious material.

The reader may find the book unsatisfying because so few definite answers can be given; but this is due to our present state of knowledge of the subject, and Dr. Marcuse is to be congratulated on not attempting to fill in the gaps where there is no clear evidence. All in all, this little book provides the layman with a well-balanced account in a field which is still the happy hunting ground of the quack and the charlatan.

The Year Book of Urology (1958-1959 Year Book Series). Edited by William Wallace Scott, M.D., Ph.D.; 1959. Chicago: The Year Book Publishers. Sydney: W. Ramsay (Surgical) Limited. 7½" × 5", pp. 368, with 82 illustrations. Price: £4 2s. 6d.

THIS volume is introduced by a report on the eleventh congress of the International Society of Urology, held at Stockholm, contributed by J. A. Campbell, which includes an interesting account of a visit to Leningrad and Moscow after the congress. The selected abstracts which form the main body of the book are arranged in the usual sections. The inclusion of a full section on the adrenal gland, and the complete omission of this heading from the current "Year Book of General Surgery', suggest that in America this field is now firmly regarded as being a part of the urologist's province. Phæochromocytoma continues to command considerable attention, with the inclusion of abstracts of nine papers on this topic. In the section on the ureter, eight abstracts discuss the use of sections of the intestine for diversion of the urine. In that on the bladder, the evergreen question of nocturnal enuresis receives three mentions. Of more importance, four papers are noticed which discuss procedures for dealing with exstrophy of the bladder; three of these concern techniques for repair and closure of the exstrophied bladder; one is a report of the long-term results in four cases in which the old-fashioned procedure of uretero-sigmoidostomy and cystectomy had been performed. Of these four patients, one was killed in a motor accident almost 25 years after the uretero-sigmoidostomy had been carried out at the age of 12 years; at autopsy the kidneys and ureter appeared normal, and there was no histological evidence of any pathological involvement of the renal parenchyma. In another case, diversion into an isolated ileal loop was undertaken twenty years after the original operation at the age of two and a half years. The section on the prostate includes the usual crop of abstracts of papers on various methods of prostatectomy; that on the genitalia includes abstracts of seven papers on hypospadias, and of ten on tumours of the testis. This is, of course, only a small selection of the variety of topics covered in this volume; like other members of the Year Book family, it is one which will be perused with interest both by specialists in this field, and by many who take a more general interest in the subject.

Books Received.

[*The mention of a book in this column does not imply that no review will appear in a subsequent issue.*]

"An Introduction to Genetics", by C. M. M. Begg, M.A., B.Sc., Ph.D.; 1960. London: The English Universities Press, Limited. Melbourne: G. Malcolm Titt. 9¾" × 7", pp. 304, with 122 illustrations. Price: 59s. 3d.

"Clinical Toxicology", by C. J. Polson, M.D. (Birm.), F.R.C.P., M.R.C.S., and R. N. Tattersall, O.B.E., M.D. (Lond.), F.R.C.P.; 1960. London: The English Universities Press, Limited. Melbourne: G. Malcolm Titt. 8¾" × 5¾", pp. 604. Price: 68s.

"Progress in the Biological Sciences in Relation to Dermatology", edited by Arthur Rook, M.D., M.R.C.P.; 1960. Cambridge: University Press. 9¾" × 6", pp. 496, with illustrations. Price: 84s. (English).

"The 20th Century and Your Heart: Arteriosclerosis, Angina Pectoris, Coronary Thrombosis", by Henry J. Speedby, M.D.; 1960. London: Centaur Press, Ltd. 8½" × 5½", pp. 192. Price: 18s. (English).

"Textbook of Gynæcology", by J. H. Peel, M.A., B.M., B.Ch. (Oxon.), F.R.C.S., F.R.C.O.G.; fifth edition; 1960. London: William Heinemann, Medical Books, Ltd. 8½" × 5½", pp. 518, with 209 illustrations. Price: 30s. (English).

"Medical Terminology for Radiographers", by Paul M. Davies, M.B., B.S., D.P.H., F.F.R., D.M.R., with a foreword by Professor George J. Cunningham, M.B.E., M.D.; 1960. London: William Heinemann, Medical Books, Ltd. 7¼" × 4¾", pp. 244. Price: 15s. (English).

The Medical Journal of Australia

SATURDAY, MAY 21, 1960.

STARTING THE RIGHT WAY.

AFTER the meeting of the Federal Council in February of this year, the General Secretary, Dr. J. G. Hunter, wrote and advised the Parent Body of the British Medical Association that the Federal Council had decided to proceed with the formation of an Australian Medical Association. The following reply, addressed to the President of the Federal Council, Dr. H. C. Colville, has now been received from the Chairman of the Council of the Parent Body, Dr. S. Wand:

Dear Dr. Colville,

I am writing to let you know that the Council of the Association, at its meeting last week, had before it Dr. Hunter's letter of 8th March containing the news that it had been decided to go ahead with the formation of a Medical Association of Australia.

I think that most of us realised that a time would come when you would feel that a separate Association should be launched, but I want you to know how anxious we in this country are that there should be no loosening of the very close bonds which have always existed between doctors in both countries.

It is for this reason we so warmly welcome your wish that the new Association, when formed, should be affiliated with the parent body.

May I therefore, on behalf of the Council here, thank you and the Federal Council for its loyal support of the Association in the past, and extend our warmest good wishes for the future.

Yours sincerely,
(Sgd.) S. WAND,
Chairman of Council.

On receipt of this letter Dr. Colville replied in the following terms:

Dear Dr. Wand,

I wish to acknowledge and thank you for your letter conveying the formal approval of the Council of the British Medical Association of our intention to proceed with the formation of an Australian Medical Association.

It is most heartening to me to receive the blessing and good wishes of your Council, and I can assure you that when an appropriate stage is reached, we will set in motion the machinery for achieving the closest possible affiliation with the Home Association, which I am sure will be in a form that will bring about a permanent bond between members of the medical profession in our two countries.

With kindest regards,
Yours sincerely,
(Sgd.) H. CECIL COLVILLE,
President.

This correspondence will, we are sure, be viewed with the greatest satisfaction by members of the medical profession in Australia. It has been fortunate for us that the British Medical Association in Australia has had within its membership virtually all the practising doctors in Australia, and it is the Federal Council's hope that, by consultation with all sections of the profession in Australia, it will be able to continue and strengthen this state of affairs in the new association on a generally acceptable basis. Most people will agree that this is a right and desirable objective. The Parent Association has never enjoyed quite this happy situation, and the profession in the United Kingdom has suffered because of its disunity, but we in Australia owe a very real debt to the Parent Association and will want to recognize it in the future. Whatever the form of our new association, its affiliation with the British Medical Association will be generally approved and, indeed, expected in Australia.

"A DECENT BLOKE."

IN 1931 a young doctor reluctantly accepted a position with the Northern Territory Medical Service. It was not his wish to go to the Territory, and he tells us frankly that he went by compulsion; but when after three years he returned to the lights of a capital city, it was with real regret. Relating this experience in an entertaining little book recently published,[1] Dr. F. B. McCann says that on looking back he could see how fortunate he was to have had the opportunity of living and working in the Territory. Not that it was attractive country. It was the real outback, undeveloped and, indeed, primitive. It was somewhat of a task, he tells us, to discover what kept Darwin going, for there was really no trade or industry worth mentioning. "The pastoral industry was dead, the meat-works were closed, and while sporadic attempts had been made to start agriculture, all that had happened can best be described by the old-timer's story of how the government, after spending thousands of pounds on an experimental farm, eventually produced one pumpkin, which 'some cow pinched'." The one thing of which the place could be honestly proud was its terrific thirst. Katherine, at which he was stationed for some time, with his wife and small daughter, was "one of the most cheerless townships imaginable".

> It consisted, in order of importance, of two pubs, a police station, a post office, a store, a few ramshackle houses, and what was called a "hospital". In the dry season it was a dust-bowl and in the wet a sea of mud, from which in the absence of roads, it was impossible to escape except by train. My residence was a tin shed with a dirt floor. It provided shelter not only for us, but also for the motor-car.

Even in Alice Springs, where conditions were somewhat better, he had a practice "extending for nearly a thousand miles north and south, and as far as I liked east and west". Calls from four or five hundred miles away were not uncommon, and tracks were primitive. In those days aeroplanes were rare.

[1] "Medicine Man", by Frank McCann; illustrations by Phil Taylor; 1959. Sydney: Angus & Robertson, Limited. 7¾" x 5", pp. 216, with many illustrations. Price: 17s. 6d.

A doctor's life in the Territory was neither easy nor luxurious. What then was there to make the difference between a reluctant arrival and a reluctant departure? It was apparently the comradeship and the genuine sense of values which often prevail in places where the varnish is off. In such places, McCann points out, artificialities count very little, and one learns to judge people not by false standards of birth and wealth; the assessment is made solely on whether or not a man is "a decent bloke". A man's past history is not counted against him, provided he measures up to the prevailing standard. There was no exemption to this rule in the Territory, even for doctors. However privileged they were in other parts of Australia by reason of their "magic title", McCann comments, in the Territory they had to stand the test as to whether they were real men; if they failed, they were let know in no uncertain way. After all, he continues, social barriers did not amount to much in a land where you did not know who was to be your rescuer should you be in danger of perishing on a waterless track. Maybe it would be someone just released from gaol. This spirit of true mateship is, he suggests, the real explanation of the attraction of such places as the Territory.

Most of Dr. McCann's book is taken up with anecdotes of greater or less importance, many of them amusing, a few tragic. It is not concerned only with the Territory, but the best stories and the most significant happenings and observations relate to the three years spent there. Although not a literary masterpiece, the book is readable and unpretentious and real. We can laugh with the author as he describes the unorthodox justice of the outback and the many roles the medical officer had to play as justice was administered, or the casual behaviour of the bushman who admitted himself to hospital, prescribed his own treatment and discharged himself, or the doctor's pursuit of a naked lubra in the course of his professional duties. We can sympathize with the young doctor when a child with intussusception, operated on under the most primitive conditions, died. We can share his admiration for the intrepid flying doctor Clyde Fenton, whose exploits have become legendary, and for missionaries and others who live dedicated lives under difficult conditions. There are stories to suit all tastes, but behind them all are frankness, humour and warmth. The author's appreciation of worthwhile values will appeal to most of his colleagues, for the simple fundamental standard by which we are told a man, including the doctor, is assessed in the Territory is, and always has been, at the heart of medical ethics. With current changes in both medicine and society, with striking advances in medical knowledge accompanied paradoxically by a good deal of debunking of the magic powers of the doctor, the medical profession faces a stern challenge. It can and should emerge from this with greater respect and trust than ever; but it will fail miserably if it ever forgets that, whatever else he may be, a doctor is expected to be "a decent bloke".

Current Comment.

SEWAGE CONTAMINATION OF BATHING BEACHES.

IN spite of a discouraging climate, the English were among the first to popularize sea-bathing as a holiday pastime. They were also pioneers of sewage disposal, but in coastal regions it was deemed adequate to discharge sewage directly into the sea at properly selected outfalls. In the years since these patterns first emerged, both the numbers of people using the beaches and the amount of sewage being discharged have enormously increased. On many parts of the coast every available beach is now a bathing resort during the holiday season, and sewage outfalls which were once comfortably remote may now be embarrassingly close to populous bathing spots. This situation has from time to time caused concern to those responsible for the people's health, and on one recent occasion the *British Medical Journal* remarked that "England is fast becoming a jewel set in a sea of sewage".[1] However, this is but a particular case of a world-wide problem, for at all modern coastal cities the sea serves two conflicting purposes, one recreational, the other as a place for the disposal of refuse, particularly sewage. This presents a recurrent headache to public health officers. The most popular beaches are naturally those nearest to cities, which are inevitably those nearest the sewage outfalls. Because of this concentration of the problem at the centres of population, in spite of her enormous coastline Australia is no stranger to the question, and a memorandum on this topic recently published by the British Medical Research Council[2] is of general interest. This memorandum embodies the results of the researches of a committee set up in 1953 by the Public Health Laboratory Service to investigate the whole problem of the sewage contamination of bathing beaches. The committee found that there was little previous work on the subject to guide them, and the extensive bacteriological investigations undertaken will be of considerable interest to those concerned with this aspect of public health. However, the committee's conclusions are mainly negative. Bacteriological evidence of sewage contamination was readily available, but in the worst cases examined, this was already painfully obvious to the naked eye. Coliform counts at any one beach varied enormously depending on such factors as the state of the tide. The committee considered that it was unrealistic and impracticable to think in terms of the standards of purity expected in drinking water, and that no other bacteriological standard had any real meaning in such circumstances. It is suggested that it is a waste of time to take occasional samples of sea-water and submit them for a fæcal coli count. "If it is already known that sewage is being discharged on to the beach concerned, the laboratory finding does not provide much further enlightenment." The committee's memorandum emphasizes the rapid dilution which takes place when sewage is discharged into the sea, an effect which is enhanced by the fact that sewage organisms rapidly disappear from sea water owing to various factors other than mere dilution; under experimental conditions, this "disinfectant action" of sea water accelerates as the temperature rises. Conversely, most intestinal pathogens are infective only when ingested in considerable numbers, and at dilutions ordinarily encountered it would be necessary to imbibe very large quantities of sea water to take an infective dose. Typhoid fever is of course an outstanding exception to this rule and in this case infection may be caused by the ingestion of a relatively small number of organisms.

Investigation of the epidemiological aspect of the problem also presents great difficulties. However, the few published reports of investigations bearing on the question have failed to reveal any serious public health risk from bathing in contaminated waters, and the com-

[1] Quoted in *The Times*, July 13, 1959.

[2] Medical Research Council Memorandum No. 37, "Sewage Contamination of Beaches in England and Wales", 1959. London: H.M. Stationery Office. Price: 2s. 6d. (English).

mittee found no evidence of serious outbreaks of disease which could be associated with bathing, even on beaches where sewage contamination was gross and obvious. Special attention was devoted to two conditions, poliomyelitis and enteric fever. Comparison of the histories in cases of poliomyelitis occurring among seaside residents with an appropriate control series plainly indicated that bathing history was irrelevant in the causation of the disease. Enteric fevers (i.e. typhoid and paratyphoid fevers) are good subjects for epidemiological investigation because the infecting organisms are readily isolated and typed, but the committee points out that the incidence of such fevers has been so low in England and Wales during recent years that an investigation similar to that undertaken in the case of poliomyelitis has not been feasible. All known cases of enteric fever are investigated by the Enteric Reference Bureau, and in recent years these investigations have detected less than one case of enteric fever per year with a history pointing to a possible connexion with sea bathing. Four such cases occurred in 1956-1958, and these were associated with two of the beaches studied by the committee in which sewage contamination was very bad. The infecting strains of the paratyphoid bacillus involved had also been isolated from the water concerned, but the committee takes the view that, in view of the extreme dilution of the organisms in sea water, it is far more likely that the patients in these cases were infected by direct contact with undisintegrated fæcal matter than by swallowing contaminated sea water. At some of the beaches concerned the sewage is treated by comminution—that is, by mechanical disintegration—before discharge into the sea. The committee states that bacteriological results so far obtained have not shown that this has any obvious effect on the topographical distribution of pollution. However, it is conceded that such treatment may be desirable on health grounds, as well as for æsthetic reasons, because it would reduce the chances of contact with a heavy concentration of excreta from carriers, and would increase the exposure of organisms to the disinfectant action of sea-water.

In the concluding remarks of this memorandum, the committee suggests that "with the possible exception of a few æsthetically revolting beaches round the coasts of England and Wales, the risk to health of bathing in sewage-contaminated sea-water can, for all practical purposes, be ignored". It is pointed out that pathogenic organisms isolated from sea-water are an indication of infection already present in the neighbouring community, and that in assessing the risks to health of bathing in a given area, the local record of gastro-intestinal disease during the relevant period should be borne in mind. It is urged that from the public health point of view it would be more profitable to concentrate on immediate environmental risks such as those of food poisoning, rather than on "the remote risk of infection with the same food poisoning organisms after they have passed down the sewer outfall".

However, to many public health officers the extent to which the risk of beach contamination has been played down in the Medical Research Council's memorandum will appear excessive. It is perhaps comforting to know that we can bathe in sewage-contaminated sea-water with little risk, but apart from æsthetic considerations modern standards of preventive medicine demand that this risk should be reduced to an absolute minimum. That the risk of sewage contamination of ocean beaches is not entirely negligible is vividly illustrated by the recent example of the City Beach in Western Australia, briefly referred to by D. J. R. Snow in a recent issue of this Journal[3] and outlined more fully in his annual report for 1958.[4] In this case at least 10 out of 15 cases of typhoid fever which occurred early in 1958 were associated with the City Beach, at a time when contamination of the beach was known to be gross, and no further cases occurred after bathing from this beach was prohibited. (Dr. Snow informs us that since then steps have been taken to improve the safety of the effluent before it reaches the sea, and the outfall pipe has been extended to a point 3000 feet off shore—instead of 300). An interesting point about this outbreak is that five different strains of *Salmonella typhi* were isolated from the victims. This was taken to show that the outbreak was caused by the pooled organisms from several ultimate sources, which would tend to incriminate sewage rather than individual carriers. It is not unlikely that the number of typhoid carriers in parts of Australia is at present unusually high, owing to the presence of a large number of recent immigrants, many of them from countries where typhoid fever is still relatively common. It may be that the English owe their immunity to any obvious ill-effects from the contamination of their beaches to the rarity of typhoid carriers among the population of Britain.

[3] MED. J. AUST., 1960, 1: 632 (April 16).

[4] Report of the Director of Epidemiology in the Annual Report of the Commissioner of Health, Western Australia, for the year 1958 (in the press).

MEDICATED SALT IN MALARIA PROPHYLAXIS.

DURING the coming months tribal people living in isolated mountains in Cambodia, Netherlands New Guinea and the Philippines will be taking active part in the Worldwide Malaria Eradication Campaign sponsored by the World Health Organization. The mountain tribes which will be cooperating with national health authorities and WHO experts live in heavily malaria-infested areas where serious difficulties have been encountered in attempting to interrupt malaria transmission by spraying with residual insecticides. To overcome these difficulties experiments will be made with Pinotti's method of adding anti-malaria drugs to salt, in the same way as iodine is added to combat endemic goitre. This idea was originally conceived by Pinotti and developed in collaboration with E. Palini, chief of the laboratory of the National Institute of Endemic Diseases in Brazil. They selected chloroquine as the drug of choice, and their method has been applied on a large scale and with considerable success in that country. However, before the method can be applied on a mass scale in new territories, pilot trials are necessary to discover the pitfalls of a different environment, and to determine the best anti-malaria drug for the purpose.

These pilot trials with medicated salt have already begun in Cambodia and New Guinea and are expected to start later this year on the island of Mindoro in the Philippines. The full cooperation of the people living in the test area is needed if the experiments are to become a success. During the census preceding the experiments, the malaria experts have made certain that this cooperation is forthcoming. In the test area of Pailin in north-western Cambodia, for example, the inhabitants at their own expense have helped to make the tests possible by converting jungle trails into roads and bridging rivers by means of logs lashed on to large stones. The malaria teams can now get in by car and truck, the first motor vehicles ever to arrive in the area.

The medicated salt used in Cambodia will be mixed at the Pasteur Institute in Phnom-Penh, capital of Cambodia. It will then be packed in plastic bags, sealed and brought by truck to the test area. Once a month every family will receive, free of charge, two kilograms of medicated salt. It does not look or taste different from the salt they have always used. But it contains enough anti-malaria drug to kill the malaria parasites within the human body. In time there will be no more parasites for the mosquitos to transmit. The medicated salt will be distributed under constant supervision, and at regular intervals during the experiments blood tests will be made to check how efficient the method is. The experiments with medicated salt in the three areas of the Western Pacific are expected to last for one year. It is likely that they will result in one more powerful and precise mass technique for combating malaria.

Abstracts from Medical Literature.

SURGERY.

Proximal Colostomy for Gastro-Jejuno-Colic Fistula.

F. R. C. JOHNSTONE (*Arch. Surg.*, March, 1959) reviews 24 cases of gastro-colic and jejuno-colic fistula occurring in 21 patients after surgery for peptic ulceration. The most important symptom in these patients is the onset of diarrhœa. The author states that such patients should have an early barium enema X-ray examination, and that this confirms the diagnosis in all cases in which it is employed. The best results of treatment were obtained by early single stage operation to resect the fistula, and to reduce the possibility of recurrent ulcer by adequate partial gastrectomy, with vagotomy if necessary, in those patients who were fit for such a procedure. Proximal colostomy was found to benefit only those whose fistula was a jejuno-colic one, whereas patients with a gastro-colic fistula were not benefited. Gastro-colic fistula was best revealed by barium meal X-ray examination.

Obstruction of the Small Intestine with Acute Circulatory Failure.

J. DEVAMBEZ AND M. LEROY (*Presse méd.*, May 16, 1959) report a case of obstruction of the small intestine, in which the patient appeared moribund on her admission to hospital because of the sudden onset of acute peripheral circulatory failure. The complete absence of pulse and of blood pressure brought about anuria which lasted for three and a half hours, but no cerebral disturbance except slight polypnœa. Resuscitation along classical lines produced no amelioration of the patient's condition, and it was decided that the only hope of saving her life lay in removing the obstruction. Operation was accordingly undertaken, and as soon as the obstruction was relieved, noradrenaline was given. The patient's condition immediately improved, and she made a complete recovery. The authors stress their belief that noradrenaline is capable of curing even the gravest circulatory failure, since, after a period of four and a half hours in which she had no perceptible pulse and no blood pressure and passed no urine, the patient recovered without a trace of cerebral or renal damage.

Experimental Production of Acute Hæmorrhagic Pancreatitis.

R. M. HARDWAY AND D. G. MCKAY (*Surgery*, April, 1959) show that it is possible to produce acute pancreatic necrosis in dogs by means of an intra-aortic injection of incompatible human blood. This reaction is associated with an episode of intravascular clotting which occurs in the pancreas as well as other organs. They state that small quantities of incompatible blood are apparently more potent in causing pancreatic lesions than are larger quantities. The authors conclude from this study that intravascular thrombi in the pancreas will produce focal aseptic hæmorrhagic necrosis and the pathological picture of acute hæmorrhagic pancreatitis.

Thrombocytopenia in Massive Transfusion.

S. GOLLUB *et alii* (*Surgery*, March, 1959) present evidence which indicates that simple thrombocytopenia is not the cause for the abnormal bleeding seen after massive transfusions in the patients studied. They think, however, that it is possible that the low platelet counts might have been contributory in producing the hæmorrhagic diathesis observed. They consider that a large part of the thrombocytopenia seen in the patients studied was probably due to dilution of the patient's blood with platelet-poor banked blood. They consider that it is difficult to establish simple thrombocytopenia as the cause of any abnormal bleeding tendency, especially after massive transfusions.

Thrombocytopenia and Hypocalcæmia.

H. S. WINCHELL, S. GOLLUB *et alii* (*Surgery*, March, 1959), as a result of their studies in dogs on the production of thrombocytopenia and hypocalcæmia, conclude that the incrimination of either of these conditions as a sole cause of a hæmorrhagic diathesis is not warranted.

Traumatic Arterial Lesions and Cerebral Thrombosis.

E. A. LINELL AND MARY I. TOM (*Canad. med. Ass. J.*, November 15, 1959) report in detail nine cases of traumatic cerebral arterial lesions causing vascular occlusion and cerebral softening. The vessels were damaged by indirect violence resulting from head and neck injuries. The authors stress the vulnerability of the internal carotid and middle cerebral arteries to indirect trauma. The intima is usually damaged, with rupture and recoil of the internal elastic lamina, predisposing to secondary thrombosis.

Prophylactic Cholecystectomy.

J. LUND (*Ann. Surg.*, February, 1960) has studied the natural history of cholelithiasis in 526 patients followed for five to 20 years. This study showed that, irrespective of the clinical severity, at least one-third to one-half of the patients subsequently develop severe symptoms and/or complications. Complications (acute cholecystitis, common duct stones) occur later in at least one-quarter of the patients. The prognosis for patients with asymptomatic stones, according to previous publications and the present series (34 cases), is approximately like that in other types of case. The mortality of cholelithiasis proved to be at least 2·7%. However, a number of deaths had been avoided by operation. There is a marked increase in mortality with advancing age. Over the age of 60 years, there was a minimum mortality of 7·2%. The risk of developing cancer of the gall-bladder in the presence of cholelithiasis was found to be at the most 1%; three such cases occurred in the present series. Prophylactic removal of the gall-bladder was considered to be indicated in all gall-stone patients who were reasonably good surgical risks. This will prevent much future morbidity. With the present low mortality rate of elective cholecystectomy, primary operation is likely to carry a lower mortality than non-operative treatment. The risk of carcinoma of the gall-bladder is no higher than the operative mortality in elective cholecystectomy. Thus, the risk of cancer, *per se*, does not indicate prophylactic cholecystectomy, but it intensifies the indication for operation, since at present the only effective measure against gall-bladder carcinoma is prevention.

Hæmorrhagic Disorders Associated with Surgical Operations.

W. S. LORIMER (*Ann. Surg.*, January, 1960) discusses the causes and treatment of excessive hæmorrhage in surgical patients and reports six illustrative cases. Acquired disorders are more common than congenital ones as causes of the acute hæmorrhagic diathesis. These may be the result of therapy or they may occur in a wide variety of diseases. Fibrinolysis is probably the commonest cause of the unexplained bleeding diathesis following operation. A simple scheme is given for orderly diagnosis involving complete blood count, clot studies (retraction and stability), bleeding time, platelet count and prothrombin time. If these are normal, the prothrombin consumption test is performed to rule out plasma factor deficiency state. Preferably, although this is impracticable at times, these tests should be performed prior to the administration of blood. The history of a bleeding tendency is highly significant and is more important than routine screening tests. The treatment is to maintain adequate blood volume and supply the deficient factor. Fresh whole blood is indicated in all cases including those of hæmophilia and other plasma factor deficiencies. Additional specific agents which may be required are 50 to 150 mg. of vitamin K_1 oxide, administered as a slow intravenous infusion for hypoprothrombinæmia, ACTH for platelet deficiencies, 2 to 10 grammes of fibrinogen given intravenously for afibrinogenæmia and fibrinolysis, and œstrogenic substance (20 to 80 mg.) given intravenously for fibrinolysis, especially in cases of prostatic carcinoma.

Heart Excision and Replacements.

M. H. CASS AND SIR RUSSELL BROCK (*Guy's Hosp. Rep.*, Numbers 2 and 3, 1959) report experiments conducted to explore the possibility of transplantation of a heart from one dog to another. They point out that the problems to be faced are, first, the biological one of the ability of a homotransplant to survive more than a short time, and second, the technical problem. They state that the first may be insuperable, but that for the moment they are concerned only with the second. They note that several authors have described heart transplantation in which the donor heart is attached to the neck vessels of the recipient; however, their report describes the technique employed in attempts to excise the heart and replace it in its normal site. The authors describe six different attempts, each of which varied in some important detail from the others. In all a heart-lung by-pass machine was used to maintain the circulation during the operation. In the first five attempts

the heart was dissected out of its bed, with division of all the arterial and venous channels, then sutured back in position. In the sixth experiment the heart was removed from one animal and sutured back into position in another. The authors describe the technical difficulties encountered. In every case complete success was made impossible by failure to control bleeding from one or other of the suture lines. However, it did prove possible to maintain the circulation for a time with the replaced heart. After cardiac arrest and maintenance of the circulation by a pump-oxygenator for two hours, it was possible to restore regular effective cardiac contractions, capable of maintaining a pressure of 80 mm. of mercury for one hour.

Late Results of Aortic Homografts.

L. O. SHERANIAN, J. E. EDWARDS AND J. W. KIRKLIN (*Surg. Gynec. Obstet.*, September, 1959) report the results of a survey of the late results in 110 patients with abdominal aortic aneurysms who were treated by resectional placement of aortic homografts. These patients were operated on at the Mayo Clinic between May, 1953, and May, 1957. Out of 135 patients with abdominal aortic aneurysms who underwent surgical exploration during this period, in 10 cases the lesion was considered inoperable, in 15 the aneurysm was resected and replaced by a synthetic graft, and in 110 an aortic homograft was used to replace the excised vessel. Of these 110 cases, in 104 the operation was elective and in six the operation was an emergency one, the aneurysm having already ruptured. There were 18 deaths in hospital, three of them being of patients who underwent emergency operation; the over-all operative survival rate was therefore 83·6%. Eight of the deaths in hospital were due to post-operative hæmorrhage related to the homograft; five of them were due to rupture of the wall of the homograft and three to hæmorrhage apparently from the suture line. Of the patients who left hospital, one was lost to follow-up, and of the remaining 91, eight had died during the period under review, three of them as a result of failure of the homograft, at intervals of 18 months to two and a half years after operation. In each of these three cases death was caused by uncontrollable gastro-intestinal hæmorrhage, due to rupture of the homograft into the retroperitoneal part of the duodenum. In conclusion, the authors state that the study was undertaken to determine whether aortic resection and placement of a homograft in cases of abdominal aortic aneurysm are justified. They find that the survival rate of patients with abdominal aortic aneurysms treated by resection and grafting is better than that of untreated patients. They suggest that the use of plastic prostheses may prove advantageous in avoiding the problem of graft failure which may occur when homografts are used.

Surgical Treatment of Aortic Stenosis.

R. J. BAIRD AND W. G. BIGELOW (*Canad. med. Ass. J.*, November 15, 1959) review their experiences in the surgical treatment of aortic stenosis. They report on 65 patients operated on between 1953 and 1959. Fifty-nine operations were performed by the transventricular route and six patients were operated on under direct vision. The authors, reviewing the natural history of aortic stenosis, stress the fact that from the onset of first symptoms such as syncope, angina or heart failure, 50% of patients suffering from aortic stenosis will be found to be dead within two years. In a follow-up examination of their surgically treated patients after intervals varying from six months to six years, they found that 80% of those who left hospital were alive with moderate to marked clinical improvement. The authors stress that the results of surgery, although by no means perfect, are very worthwhile in a large percentage of cases.

Diaphragmatic Hernia through Congenital Defects.

J. A. W. BINGHAM (*Brit. J. Surg.*, July, 1959) discusses herniation through congenital diaphragmatic defects and points out that the results of surgical treatment have improved during recent years. He reviews 14 of his own cases to indicate the surgical management. Neither eventration of the diaphragm nor herniation through the œsophageal hiatus is included. Three types are recognized: (i) postero-lateral defects; (ii) retrosternal defects; (iii) extensive defects involving most of the hemidiaphragm. Postero-lateral defects lie at the point of attachment of the diaphragm to the eleventh rib or close to that point and occur most commonly on the left side; in infants these are generally small, 2 or 3 cm. in diameter. In the great majority of postero-lateral defects there is free communication between the peritoneal and pleural cavities, less often there is a thin hernial sac. Congenital retrosternal hernias are less common and almost invariably extend into the anterior mediastinum; these probably always possess a hernial sac. Defects of the third type are regarded by some as severe forms of the postero-lateral defect, but the author prefers to regard them as a separate type. The postero-lateral hernia may constitute a surgical emergency in the early neonatal period. The symptoms and signs and the radiological appearances are discussed and the author recommends a plan of surgical management and technique. The author states that the thoraco-abdominal approach which he describes has proved very satisfactory. As regards retrosternal defects, he has employed an epigastric approach in three cases and found it to be satisfactory.

Suction in the Control of Cavities and Fistulæ.

K. Q. VANDENBOS, C. W. HUGHES AND W. F. BOWERS (*Arch. Surg.*, June, 1959) point out that cavities, tracts and fistulæ tend to close spontaneously if kept clean and dry, providing that there are no foreign bodies, neoplasms or granulomatous infections present. Suction applied through a catheter through a wound, cavity or tract with saline irrigation will keep such wounds clean and dry so that uncomplicated healing follows quickly. It is pointed out that these problems are not bacterial, and antibiotics need not be used. The authors have used continuous suction under skin flaps formed in such undermining operations as radical mastectomy, radical neck dissection, axillary and inguinal dissections, and in the subcutaneous area after incisional hernia repair and the perineum after abdomino-perineal resection. They point out that such wounds heal rapidly and well, only a small dressing being needed. They state that no wound infections have eventuated from the use of the catheters. Continuous suction has also been used by the authors in intra-abdominal abscess cavities and liver abscess cavities, with periodic saline irrigation to promote cleanliness. Such cavities have healed rapidly and well without antibiotics. The authors also report the use of continuous suction in cases of anastomotic leaks or fistulous tracts from œsophago-gastric junction, duodenal stump or injury sites, and of pancreatic fistula and fæcal fistula. Rapid healing eventuated in such instances without abscess formation or other complications, without the need for surgical intervention and without the use of antibiotics.

Surgical Treatment and Pathogenesis of Stasis Ulcers.

M. N. ANDERSON AND J. G. STEPHENS (*Ann. Surg.*, July, 1959) present a controlled study of the treatment of stasis ulceration of the legs by excision and skin grafting. Stasis ulcers were treated by grafting in 20 patients, after excision. Seventeen obtained a good result, two a fair result, and one a poor result. There was no instance of recurrent ulceration in a follow-up period of two to three years. In 15 patients with ulcers who were treated conservatively for the same period, there were 10 instances of recurrent ulceration. In venographic studies of 20 extremities, only three had normal deep veins, confirming the impression that the majority of stasis ulcers are produced by deep venous disease and uncommonly result from uncomplicated superficial varicose veins. Deep venous obstruction appeared to be less favourable than recanalization of a previously thrombosed vein.

Post-Amputation Radiographic Evidence for Small Artery Obstruction in Arteriosclerosis.

E. A. EDWARDS (*Ann. Surg.*, August, 1959) states that in spite of the general assertion that arteriosclerosis does not affect small arteries in non-diabetic subjects, it is commonplace to find occlusion of both named and unnamed small arteries and arterioles in toes or feet amputated from such individuals. The author studied 13 arteriosclerotic lower limbs after amputation, by the method of angiography. He shows that the smaller named and unnamed arteries of the foot are the sites of extensive occlusion in arteriosclerotic limbs which come to amputation. The arteries involved include the branches to both the medial and lateral parts of the heel, as well as the metatarsal arteries and those to the free borders of the first and fifth toes. The other digital arteries were less frequently diseased. No difference was found in the pattern of occlusion between the feet of diabetic and non-diabetic subjects, and occlusions were as widespread in most of the non-diabetic subjects as in the diabetic.

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on January 21, 1958. PROFESSOR W. K. INGLIS was in the chair. The principal speaker was DR. K. R. DAYMOND, Honorary Assistant Orthopædic Surgeon.

Clinical History.

CASE I.—About two months before the admission to hospital of a boy aged six years, his mother noticed, when putting on his sock, that there was a tender swelling above his right ankle. A short time later he complained of pain in that area and she noticed that when he played with other children he dragged his right leg. The swelling increased in size, extending downwards towards his foot, and grew more tender. It had not been red at any time, and the pain did not wake him at night. He had had no recent infections, but had suffered in the past from bronchitis, but not from asthma. He had had no recent attacks of bronchitis. He had also had measles and mumps and had received immunization against diphtheria. His parents were young and healthy, and his sister, aged two years and nine months, had been treated for iron deficiency anæmia.

On examination of the patient, there was a hot, tender swelling on the "inner aspect of the right tibia". The other bones were normal. No abnormality was found in the cardiovascular, respiratory or digestive systems. An X-ray film showed a large transradiant area on the lateral aspect of the right tibia, just above the epiphyseal line. The radiologist's opinion was that the appearances strongly suggested a secondary neoplasm, and he requested information regarding a renal primary tumour and also suggested that intravenous pyelography be performed.

The boy was admitted to hospital, and a biopsy was made on his second day in hospital. The surgeon remarked that such of the lesion as was removed shelled out easily. During the first week the patient's temperature rose to 99° F. every day; after that the temperature was normal.

CASE II.—A boy, aged three years, was admitted to hospital with symptoms referable to his right arm. Six months previously he had begun to complain of pain there, and his mother said that he seemed to be numb in that area at times. The symptoms gradually became worse, and about six weeks before his admission to hospital he was taken to his local physician, who said, after taking an X-ray picture, that there was "a cyst in the bone". He put the arm in plaster, which was removed after a month. The parents stated that there was "much more bone in the arm than before". The child did not eat well, and had failed to gain weight during the last six months. A tonsillectomy had been performed a short time before the onset of the symptoms. There were no other symptoms. He had had no other illnesses or operations. He had been fully active during the six months' period except for the right upper extremity, in which his parents had noticed limitation of movement.

On examination of the patient, the right arm was tender and enlarged in diameter when compared to the left. There was doubtful enlargement of the liver, the notes stating that it was "doubtfully palpable one finger's breadth below the costal margin". No mention was made of the upper border. The other bones, the lymph nodes and all the other systems were normal. The blood pressure was 110/70 mm. of mercury.

On his admission to hospital, the child's temperature was elevated, later rising to 101° F. and not returning to normal. A biopsy was made, and after that there was still an irregular elevation of temperature at a lower level than before. Penicillin (100,000 units three times a day) was given. In spite of the fever the child was fully active and ambulant. An X-ray examination revealed a transradiant lesion in the shaft of the right humerus at the junction of the middle and lower thirds. There was very noticeable "onion-peel" appearance in that area.

Clinical Discussion.

PROFESSOR W. K. INGLIS: This afternoon we are departing to some extent from the general custom of submitting a case where there has been death and an autopsy. We have two cases of outstanding interest, and we are fortunate in having members of the Section of Orthopædics of the New South Wales Branch of the British Medical Association present. I will call on Dr. K. R. Daymond, Honorary Assistant Orthopædic Surgeon, who is the main speaker.

DR. K. R. DAYMOND: When I was asked to be principal speaker at this clinico-pathological conference, at first I was rather hesitant, because I feared the cases would be difficult to diagnose. My fears were not allayed when I learned that the Section of Orthopædics had been invited, and were confirmed when I studied the protocol and the X-rays. We have two cases for discussion, and since I feel that some of the discussion will be applicable to both cases, I intend to discuss the general features of bone lesions and point out where these features are relevant to the cases under discussion.

For those who have not seen the X-rays, I will first show the slides of these and point out the radiological appearances of significance.

The first case is a lesion in the lower end of the tibia (Figure I[1]) in a boy of six years. It is completely osteolytic, and as far as I can see there is little evidence of new bone formation, though I am not sure of this. Some spicules may be those of the old bone that remain. As far as can be seen, the epiphysis is not involved. The lesion is eccentric and involves the cortex.

The second case is that of a three-year-old boy with a lesion in the shaft of the humerus (Figure II). Situated in the centre of the shaft there is a destructive lesion with a notable amount of periosteal reaction, which has been described as onion-peel effect in the protocol. A finding that impresses me is that there is erosion of the newly formed bone, presumably an effect of the lesion. The other views show nothing further of note.

We have, therefore, two lesions in bone to deal with, and I think it will be best to treat the possibilities systematically, and I will indicate the possibilities I have considered.

Congenital.

Acquired:

(*a*) Traumatic: fracture through a simple cyst or aneurysmal bone cyst.

(*b*) Infective: subacute suppurative inflammation; tuberculosis.

(*c*) Neoplastic:

Benign: Solitary enchondroma, chondromyxoid fibroma, giant-cell tumour.

Malignant: Chondrosarcoma, osteogenic sarcoma, fibrosarcoma of bone, Ewing's tumour, leukæmia and Hodgkin's disease, reticulum-cell sarcoma, liposarcoma, œsinophilic granuloma.

Metastatic: Carcinoma and neuroblastoma.

Firstly, as regards the congenital lesions of bone, they are easily disposed of. No congenital condition can produce the distinctive appearances of either of these cases.

Trauma is not applicable to the eccentric tibial lesion. However, it can be considered in the humeral lesion if a bone cyst previously existed in this area. Bone cysts are not uncommon in children, and if a fracture occurs excessive callus tends to be formed. However, this possibility appears to be excluded by the absence of any fracture line and by the erosion of newly-formed bone. There is an equal possibility of trauma complicating an aneurysmal bone cyst in the humeral lesion. This, too, can be excluded by the absence of any remaining residue of the typical soap-bubble X-ray appearances and by the destruction of the new bone already referred to.

Infective lesions are a well known difficulty in the X-ray diagnosis of bone lesions. Most orthopædic surgeons have had the experience of clinical and radiological diagnosis of malignancy, only to find inflammation at operation. With this in mind, consideration of the tibial lesion recalls that it was noted to be hot and tender, but it was not red. The other clinical features do not especially suggest an inflammatory lesion. He had only slight fever, though such is not inconsistent with subacute osteomyelitis. In this tibial lesion I think that there is far too much cortical destruction to consider an inflammatory lesion seriously. However, in the humeral lesion the possibility is very strong. The periosteal reaction is quite characteristic, and the erosion of the new bone is certainly not inconsistent, and the clinical features are not very helpful. However, I feel that the radiological appearances show a far too uniform periosteal reaction for an ordinary subacute

[1] For Figures I, II and III, see art-paper supplement.

suppurative osteomyelitis. It would be helpful to have a blood count. I do not exclude this possibility in the humeral lesion though I do not feel that osteomyelitis is the answer.

Regarding the chronic infections, there are many possibilities, almost all remote. Of these, tuberculosis is worth considering, though itself a rare disease in bone in this country. The common form is a lesion at the end of a long bone which rapidly involves the epiphysis and extends into the joint. This form does not apply to either of these two cases, the first showing the absence of new bone formation and the second being a very unusual location. In the rarer form of tuberculosis which occurs in children, particularly those suffering from malnutrition or intercurrent disease, a cystic lesion forms in long bones, and they may be multiple, in the same or other bones. The metaphysis or diaphysis is involved. The cortex is eroded and abscess forms. This cystic form could be applicable to both cases. I would like to point out that in the case of the tibial lesion we are told that there is a swelling in the region of the joint and that it is on the medial side. But we see in the X-rays that the lesion is on the lateral side of the tibia, and one wonders whether there is a generalized thickening around the joint. The second case also presents this possibility. However, on clinical grounds it appears that probably in both cases tuberculosis can be discarded. I think that the discussion so far, to my mind has satisfactorily eliminiated the possibility of congenital or inflammatory lesions, with the reservation of subacute inflammation in regard to the humeral lesion (Case II).

In discussing the neoplastic lesions, I will deal rather summarily with some, since they need little discussion; for example, simple enchondroma, which occurs in an older age group, tends to thin the cortex and is not likely to produce periosteal reaction. However, a rare simple tumour, the chondromyxoid fibroma, merits further consideration. It occurs in children, frequently in the tibia and in the metaphysis in the region of a joint. It is eccentric and erodes and destroys the cortex, and the clinical features are pain and palpable mass. It is important because of the possibility of misdiagnosis as chondrosarcoma. It is obvious from this that this is a distinct possibility in the tibial lesion, and I do not feel I can exclude this possibility without biopsy.

A giant-cell tumour needs little mention. It is rare under the age of 20 years, and there is doubt if it occurs in the metaphysis. Many tumours diagnosed as giant-cell tumours formerly are now known to be "variants" and are other tumours. In both cases under discussion the cortex is not expanded, and the epiphysis is intact.

In discussing newgrowths, the rare central chondrosarcoma can be ruled out on radiological appearances in both cases.

Osteogenic sarcoma is commoner in boys and the maximum age group is between 10 and 25 years. It is most likely to be situated at the end of a long bone, and in the tibia is commoner at the upper end, and it tends to involve the epiphysis as well as the metaphysis. Pain and swelling predominate. These features are consistent with the findings here; but secondary anæmia and loss of weight are marked in rapidly-growing lesions. There are reasons to doubt the applicability of this diagnosis to either of these lesions. The age in both is young, and in the tibial lesion the epiphysis is intact. Here also there is absence of any new-bone formation, which is rare when osteogenic sarcoma involves the cortex. I do not think that the so-called osteolytic types of osteogenic sarcoma are entirely devoid of new-bone formation as a rule. As regards the lesion in the humerus, there is new-bone formation, but the reaction, if due to osteogenic sarcoma, would be a sun-ray radiological appearance rather than the onion-peel effect noted here.

That is not to say that in rare cases these appearances may not present in cases of osteogenic sarcoma; but they would be most unusual. It would be helpful to have serum alkaline phosphatase values which we would expect to be about 20 Bodansky units. In cases in which tumour is removed the level falls, but recurs with the appearance of metastases. I think osteogenic sarcoma can be ruled out in the tibial lesion and is a very remote possibility in the second (the humeral) lesion.

There is great difference of opinion on the pathological features of fibrosarcoma of bone. In the opinion of Lichtenstein, the growth begins centrally in the bone, spreads to destroy the cortex and produces no reactive new bone. Clinically there is resemblance to osteogenic sarcoma, and these features may give rise to the suspicion of this growth; but it is difficult to be certain without biopsy. In contrast, Geschickter states that the condition begins in the periosteal or parosteal tissues and grows inwards to involve the bone. In view of these variable radiological features, one cannot entirely exclude this possibility. However, I feel that, in view of the better prognosis of fibrosarcoma over osteogenic sarcoma, we should recognize this growth and diagnose it if possible. It appears that if Lichtenstein is correct, it may explain the tibial lesion.

Ewing's tumour is difficult to discuss. The features stressed by Ewing were the age of the patient (usually between 10 and 25 years), a characteristic X-ray appearance, the gratifying response to X-ray therapy, the later appearance of lesions in other bones, and the ultimately fatal outcome. Each of these has been criticized by other writers, and there is often lack of agreement on the biopsies. Moreover, it has been suggested that cases diagnosed pathologically as Ewing's tumour have been shown to be metastatic neuroblastoma, carcinoma and lymphosarcoma.

If we accept Ewing's description and consider the lesion in Case II (humerus), we see appearances quite characteristic of it. Indeed, that was my first impression regarding it. It appears also that the entity of Ewing's tumour exists. When patients first come to hospital the growth is present usually in one bone, and that in the shaft rather than the ends. Pain is almost universal, and the neighbouring joint is stiff. In most cases a tumour is palpable, and it is tender. A daily fever to 101° F. is common, and a secondary anæmia, leucocytosis and increased erythrocyte sedimentation rate may be present. The essential and only constant radiological finding in Ewing's tumour is destruction of bone. The onion-peel effect is not often present, and if present occurs late in the disease. And it may give rise to transverse striation similar to osteogenic sarcoma, for which the radiological appearances may be mistaken.

The tibial case could represent an earlier and the humeral lesion a later case of Ewing's tumour. In both, the age group is rather low and the clinical features do not especially suggest it, and I do not feel that either of these lesions is a Ewing's tumour, though it is not possible to exclude it on the information available.

In dealing with newgrowths arising from hæmatopoietic and related tissues, we have to consider briefly firstly, leukæmia, which may involve bones—in which case multiple punched-out areas are the rule. These may have onion-peel reactive bone formation. Chloroma, which is a rare variety of leukæmic bone deposit seen in children, may also behave in a similar fashion; but the lesions are mainly in the ribs, skull and vertebræ, and long-bone involvement is very rare. Primary reticulum-cell sarcoma of bone is a growth identical in appearances with reticulum-cell sarcoma found in lymph nodes. The patients are usually adults, and the radiological appearances are those of a mottled area of rarefaction. It is a localized destructive lesion. In the case of the tibial lesion, there are some bony spicules which, though possibly reactive, I think are probably more likely areas of bone not entirely destroyed. In that case reticulum-cell sarcoma is remotely possible on radiological but not on clinical evidence. I should mention that some pathologists regard this lesion as identical with Ewing's tumour, but both surgery and X rays give better results than they do with Ewing's tumour. Bone lesions of Hodgkin's disease are variable; but this hardly needs pursuing in these cases.

Liposarcoma is very rare in bone and tends to destroy the cortex. The radiological appearances are not well enough known to merit discussion. Adamantinoma of the tibia is a neoplasm of adamantine epithelium. Someone has suggested that the only way this can occur is for the fœtus to double itself up and bite its own leg. Whatever the origin of the epidermoid epithelium may be, the growth is rare, and the patients are older than those we are discussing.

Eosinophilic granuloma is a well-known tumefactive process that characteristically simulates Ewing's tumour clinically, in that the limb is hot, swollen, and tender. It affects one or more bones. The bone is destroyed, the cortex is eroded and new bone may or may not be laid down. Fever is present at the onset, and leucocytosis may be present. The X-ray appearance may be exactly similar to the lesion in the humerus, and it merits serious consideration, since an onset at this age is not unusual. It is the probable diagnosis of the lesion in the humerus.

In concluding the discussion of the malignant newgrowths, it is only necessary to mention metastatic lesion.

Of all these possibilities age rules out most. The only possibility is metastatic neuroblastoma. In Case I (tibial lesion) the likelihood is very great that the lesion is metastatic; but the age is somewhat against it.

We should lastly mention lipoid reticuloses other than eosinophilic granuloma. None of these merits serious consideration in these cases.

A word should be said about the possibility of an adjacent soft-tissue lesion invading the bone. This appears to be most unlikely in the present cases.

I have already mentioned other investigations which may help in these cases, and in addition I have omitted to mention a Mantoux test. However, I have to make my diagnosis on the available data.

In regard to the first (tibial) lesion, I think the likely diagnosis is fibrosarcoma, though I cannot exclude Ewing's tumour, neuroblastoma or chondromyxoid fibroma.

My diagnosis in the second case is eosinophilic granuloma, though subacute osteomyelitis, tuberculosis and osteogenic sarcoma are remote possibilities. The test I would like most of all before I made up my mind would be a biopsy.

DR. H. R. T. HODGKINSON: I must say this has been an interesting discussion, but I do not agree with the conclusions. Regarding the second case (humeral lesion), I would be very surprised if it was not osteomyelitis; the X-ray picture is such as one would expect to see in an older child who had developed some resistance. It appears to be a low-grade process with a fair amount of new-bone formation. Admittedly, there is a cyst-like cavity which appears to be eroded, perhaps. But this is consistent with osteomyelitis. Also, in a growing child of this age (three years) there is often an enlargement which is visible to the mother. Ewing's tumour also always merits consideration when one sees new-bone formation, but this child is rather young to have Ewing's tumour.

The diagnosis of the tibial lesion is more difficult. The fact that the lesion was easily shelled out makes one wonder whether it was a fibroma beginning in the soft tissues and extending into the bone.

DR. W. S. L. STENING: Nothing can be added to the comprehensive differential diagnosis that we have had from Dr. Daymond. In regard to the case of the second (humeral) lesion, I take a hopeful view. The tonsillectomy a short while previously is not an uncommon occurrence before the appearance of a septic focus in bone. I am not impressed by the onion-layer appearance, which is just new-bone formation, and I think I have found several small areas, possibly sequestra, in the X-ray picture, though it is only a photograph. I think the most likely diagnosis is subacute osteomyelitis. In the other (tibial) lesion, however, I take a gloomy view. My impression is that the lesion is malignant and probably a rapidly growing one. I do not see how it can possibly be invasion of bone from a neighbouring soft-tissue tumour, because one can see islands of bone partly destroyed in the substance of the tumour. My feeling about it is that it is a secondary from a neuroblastoma.

DR. C. C. MCKELLAR: In regard to the second (humeral) lesion, my preference is for osteomyelitis, following the opinion of the last two speakers. In regard to the first case, the tibia, though it does look malignant I would like to mention two other possibilities, sarcoid and a syphilitic gumma. The X-ray picture is puzzling, because in one view I can see small fenestrations in the talus, though these are not visible in the other view. I ascribe these to photographic artefacts, though they suggest a generalized condition if they are true.

DR. H. G. MARSH: I must agree with the majority orthopædic opinion regarding the case with the lesion in the humerus, I think there are many features that suggest it is infective in origin. The X-rays do not show any significant extension into the soft tissues, and very often this is seen. I would consider this lesion a low-grade osteomyelitis.

As for the lesion in the tibia, I am as gloomy as others. For one thing, I feel sure that there is some expansion of the tibia, and this suggests that the process has been going on for some time. I am inclined to think that the tibial lesion may be an eosinophilic granuloma.

DR. A. I. RHYDDERCH: I have been struck by the fact that in cases of osteogenic sarcoma there is almost invariably a complaint of pain. Often this is so severe that the patient may beg the surgeon to amputate the limb if only to relieve the pain. I think that the humeral lesion is unlikely to be a primary bone tumour, and I lean strongly towards an infective basis—a low-grade osteomyelitis. I think that Dr. McKellar's comment is apt, and all bone tumours demand that a Wassermann reaction be done. There is often a surprise, and I have seen a case of syphilitic osteomyelitis in a child of this age.

The diagnosis of the tibial lesion is more difficult. It appears to me to be a malignant process. There is visible on the X-ray picture a soft-tissue swelling on the inner side of the leg extending out over the ankle. This was noticed by the parents. But the destruction of bone is on the lateral side towards the fibula, and suggests that there could be an extraosseous tumour involving the bone from without. The other possibility for the tibial lesion is a secondary lesion.

DR. J. D. CASHMAN: I would like to mention another possibility for the tibial lesion—that is, Letterer-Siwe disease, which is similar to eosinophilic granuloma, but carries a much worse prognosis.

Pathological Discussion.

DR. E. HIRST: The diagnosis of Case I is chondromyxoid fibroma of bone (Figure III). We owe our knowledge of this benign, rare, but important, entity to Jaffe and Lichtenstein and to Dahlin and others. They have described it as an eccentric, sharply localized area of rarefaction (sometimes traversed by trabeculæ) found in the diaphysis or epiphysis. It usually occurs in young adults, though cases have been described in the fifth decade. Two-thirds have been in the tibia near the knee.

The growth has been named for its typical histological appearance, which is that of scalloped pseudolobules separated by vascular bands of more compact growth. The lobules consist of areas of chondroid and of myxoid appearance, while the compact areas are composed of fibroblast-like spindle cells: hence, "chondromyxoid fibroma". In some sections numerous giant cells can be found, and these have led at times to the erroneous diagnosis of giant-cell tumour. The tumour is now known as one of the giant-cell tumour "variants", and this has been discussed by Dr. Daymond, who also mentioned that it is apt to be mistakenly diagnosed as chondrosarcoma. The reason for this is seen in other sections where cells appear to exhibit some degree of anaplasia.

The definitive treatment in this case was curettage, and radiological report three weeks later stated that regeneration was taking place satisfactorily. The tissue curetted weighed 8 grammes, and the growth was estimated to measure 4 by 4 by 2 cm.

In Case II the diagnosis was eosinophilic granuloma of bone. The typical appearances seen in the sections are those of the large pale cells and collections of small polymorphonuclear cells. The large cells are histiocytes, and they resemble those seen in other granulomatous lesions. The small cells are eosinophils; hence "eosinophilic granuloma". Some of the histiocytes are large, with pale cytoplasm containing lipid. Occasionally giant cells with nuclei arranged in a peripheral ring can be found. There is abundant evidence of the relationship of the commonly single punched out lesion of eosinophilic granuloma to Hand-Schüller-Christian disease and to Letterer-Siwe disease, the inclusive name of histiocytosis-X being applied. Because of the distinct possibility of transition to a multiple system disease, the prognosis of solitary eosinophilic granuloma of bone is not clear. In this case there is no evidence in the short period of observation of involvement of the pituitary gland or lungs.

Diagnosis.

	CASE I.	CASE II.
Dr. Daymond's diagnosis:	Fibrosarcoma.	Eosinophilic granuloma of bone.
Other commentators' diagnosis:	Soft-tissue fibroma involving bone.	
	Metastatic neuroblastoma.	
	Gumma.	Subacute *osteomyelitis.*
	Eosinophilic granuloma.	
	Extraosseous tumour involving bone.	

	CASE I.	CASE II.
	Letterer-Siwe disease.	
Pathological diagnosis:	Chondromyxoid fibroma of bone.	Eosinophilic granuloma of bone.

British Medical Association.

NEW SOUTH WALES BRANCH: SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on July 30, 1959, at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, DR. MUNRO ALEXANDER, the President, in the chair.

Occupational Diseases of the Chest Excluding Silicosis.

DR. ROBERT HUGHES read a paper entitled "Occupational Diseases of the Chest Excluding Silicosis" (see page 800).

DR. M. R. JOSEPH read a paper entitled "Occupational Diseases of the Chest Excluding Silicosis" (see page 804).

DR. W. E. GEORGE said that recently the association of malignant disease of the lung and of the pleura with asbestosis and with the mining of hæmatite, had been thought to be due possibly to the fact that in both cases there were large quantities of iron present in the inhaled dust. Referring to coal-workers' pneumonoconiosis, Dr. George said that he agreed entirely with what Dr. Joseph had said. In the early days of the investigation of dust diseases of the lungs, it had been thought that they were easy industrial diseases to diagnose, in view of the positive radiological evidence. In those days, radiological findings were considered of primary diagnostic importance and they were followed in importance by the industrial history and the results of clinical examination. Nowadays it was realized that the industrial history and the findings on clinical examination were, in many instances, more important than the radiological appearances. Dr. George said that he thought that, in many cases of advanced emphysema, the presence of that condition disguised or interfered with the nodular shadows one would expect to find on the X-ray film in view of the patient's industrial history and symptoms. In many cases at autopsy definite evidence of pneumonoconiosis was found to be present although there was no evidence of it radiologically during life. He thought that possibly in that condition the nodules were discharged into local emphysematous areas and coughed up by the patient. That might explain the black sputum produced long after such persons had ceased work in the coal industry. If radiographs had been taken years before the emphysemalous condition had developed, definite evidence of pneumonoconiosis would probably have been found to be present in cases of that type.

DR. COTTER HARVEY referred to the intriguing question of spontaneous pneumothorax. He said that he saw a number of patients with that condition, but in only a few cases were there possibilities of compensation. He thought that either it was becoming increasingly common, or it was more frequently diagnosed. It was not necessarily correct to assume that the lung went down like a blown-out tyre or a burst balloon; it could be a very slow leak, of which the patient was unaware for quite a time, and the doctor might be unaware for some days. When the patient consulted his doctor, the diagnosis might be pleurisy or a strained muscle or something similar, and neither might think much of it. Possibly a day or so might elapse before the doctor thought that an X-ray examination would be interesting, and then the pneumothorax would be revealed. Often the patient could not remember any physical strain causing immediate pain; the pain might be delayed and so also might the dyspnœa. The condition had been known in France for many years under the name of *pneumothorax par effort*; it commonly affected cyclists who bicycled enthusiastically over the Alps. Dr. Harvey remarked that surely that was related to effort. He himself had seen it related to swimming, surfing, and athletics. Increased intrapleural pressure must play a part, with a sudden jerk or bump or even a steady lift. He had advised men who had sustained one attack and who had to lift heavy weights in the country to breathe out, not in, and then lift. Patients were often unaware that anything had happened until they felt a funny feeling, a pain round the heart or "indigestion", and then they remembered some slight incident, such as having to push the car into the garage; but by the time they reached the "compensation" doctor the story had often become a complete drama. As Dr. Hughes had stressed, in examining such a patient it was essential to find the exact chronological sequence of events, and the story might be quite confused after it had been several times retold. Dr. Harvey finally said that it was a mystery to him how men who were heavy smokers were often able to lay the blame successfully on some industrial dust for their bronchitis, and thus obtain compensation sometimes indefinitely prolonged, while they continued to smoke and probably more than ever before.

DR. JOHN READ said that he was glad Dr. Joseph had emphasized the fact that the testing of respiratory function was more important than radiographic examination. Radiographic studies had provided much valuable information in the epidemiological and preventive fields in early years, but nowadays, in Categories I, II and III of "coal-miners' pneumonoconiosis" (radiological variety), X-ray examination had become almost a menace in diagnosis. There were two pieces of evidence for that assertion. One was provided by Cochran, who had demonstrated that there was no correlation between the X-ray findings in Categories I, II and III and a falling off in respiratory function, but that there was a good correlation between a falling off in respiratory function and the number of years spent at the coal-face among workers with no radiological abnormality. The second piece of evidence had been provided by Rivers, who had correlated histological studies of the lungs with X-ray findings among coal-workers. He had found that the grading of radiological involvement in simple coal-workers' pneumonoconiosis correlated well with the weight of coal dust in the lung at autopsy, but that X-ray grade correlated poorly with the amount of focal emphysema present. Focal emphysema, not coal dust lying in the lung, was the cause of the disability. If a man had an adequate exposure to coal dust, and one could demonstrate airway obstruction by simple physiological tests, then it was impossible to say clinically that he did not have coal-workers' pneumonoconiosis—which was a pathological, not a radiological, entity. It was not necessary that a person should have shadows in the chest X-ray film for a diagnosis of coal-workers' pneumonoconiosis to be made. The radiographic appearances did not matter unless progressive massive fibrosis was present.

DR. W. L. CALOV referred first to the mechanics of spontaneous pneumothorax; he said that they were exceedingly difficult to understand. He had tried many times to explain them on purely physical grounds, but had been unable to do so. Most of those present would remember that, on looking at a lung *post mortem*, they would see marks of the ribs on it; also, if emphysema was present, the emphysematous blebs tended to find the intercostal spaces, and to occur at the apex where the pressure was not so great. The pressure of the chest wall on the lung was not even; it was greater where the hard structures were applied to the lung, and less where the softer structures were applied to the lung. Perhaps those considerations might help to convince Dr. Hughes that effort could be responsible for rupture of one of those blebs. Dr Calov then referred to the question of pneumonia and exposure to cold. He said that it was a popular conception that one got cold and got pneumonia. In reality, a man who shivered did so because he was already ill, and not necessarily because it was very cold weather. Dr. Calov said that he had had it put to him that a man had caught pneumonia because he was exposed to extremes of heat and cold. In one case, a man was working inside a boiler in very hot conditions, and when he came out he felt extremely cold. He blamed the extremes of heat and cold for the pneumonia from which he suffered. However, when he was on his way home that night he had a shivering attack because he was getting pneumonia. The real cause was that he had exhausted himself with hard work in the boiler while he was a sick man. Perhaps if he had stayed at home in bed that day and not gone to work, he might not have got pneumonia. Referring to pneumonoconiosis, Dr. Calov said that he had examined large numbers of coal miners in the last few years. In some cases, when he looked at their X-ray films in his viewing box, he saw many shadows; when he viewed them at a window, he could see no abnormality at all. He could not decide what was the cause of the small irregular or rounded shadows in many films. He suggested that they should discard the word "nodulation" in describing X-ray appearances; it was only a shadow, and some other term should be found. He found it difficult to decide whether the shadows were due to vascular changes or to coal

fibrosis. He agreed that the degree of disability did not go *pari passu* with the X-ray appearances.

DR. K. G. OUTHRED said that one point in connexion with coal-workers' massive pneumonoconiosis had been stressed by various investigators. Although it was believed that the condition was in most cases a disease caused by both dust and the tubercle bacillus, yet those agents modified each other, and as a rule the tuberculosis did not assume the usual progress of the disease until a very late stage in life, and then less than in the general community. A few years earlier there had been a minor controversy between Dr. Fletcher and a professor of chemical engineering in Great Britain, who extracted from coal a substance called vitricin, which appeared to slow down the growth of tubercle bacilli in a Petri dish. Dr. Outhred went on to say that 50 years earlier, coal dust had been considered beneficial; maybe there was an element of truth in the idea—not like silica dust, whose relationship with tuberculosis was quite the opposite. Referring to asbestos mining in Western Australia, Dr. Outhred said that asbestos occurred in a highly siliceous matrix; it had to be separated from the quartzite in a mill. It was likely that an appreciable amount of free silica might still be in the asbestos when it arrived in New South Wales. Asbestos workers could have a mixed condition of asbestosis and silicosis.

DR. A. G. MCMANIS said that he had recently examined a patient who had been exposed to asbestos. Cavities were present in the upper lobe of the right lung, which was removed. The lobe was loaded with asbestos bodies, and the diagnosis of asbestosis was made. However, radiologically the condition had not the slightest resemblance to asbestosis. Dr. McManis asked Dr. Joseph to comment on that case. Referring to spontaneous pneumothorax, Dr. McManis said that he went all the way with Dr. Harvey in saying that in a large number of cases the patients did not know when it occurred. It was impossible to say that it occurred at rest or at any other time. He knew of six or seven people who found out that they had it only because, for some entirely unrelated reason, they had their chests radiologically examined. They never had any symptoms. That proved that one could get it without knowing anything about it. The occurrence of symptoms depended on the degree of disorganization of the chest mechanics. The condition could indubitably be related to effort. If somebody lifted a sack of potatoes and suffered pain and breathlessness, it was impossible to dodge the connexion. Further, the effort did not have to be a big one; in a case in his experience, a man got a spontaneous pneumothorax through passing flatus.

Dr. Hughes, in reply to Dr. George's remarks about iron being the carcinogenic factor, said that it might be; he did not know. It was quite posible that in a few years' time some common factor would be found to account for the various cases of lung cancer, which was not apparent at present. Dr. Harvey and Dr. McManis had both expressed fairly definite views about the onset of pneumothorax often being gradual; Dr. Hughes said that that could be so. Dr. Harvey had said that in those cases in which spontaneous pneumothorax occurred during sleep, the effort might have occurred some time before if the leak was very slow; but how could anybody know what the effort was, if effort was the cause? The patient might have been lifting some sacks of potatoes at work; on the other hand, he might have passed some flatus. Referring to the French bicycling enthusiasts, Dr. Hughes said that it was quite likely that their activity could be a factor, because while they were bicycling they would be breathing hard; that could cause a sucking action, which would be more likely to cause the blebs to burst than would actual straining. Dr. Calov had referred to a man who had been in and out of a hot boiler, developed symptoms of pneumonia on the way home and blamed the hot boiler. Dr. Hughes said that he agreed with Dr. Calov's comments. However, as the patient did not know that he was going to contract pneumonia, he could hardly stay at home and in bed. A story that was often told to him was that of, for example, a man who was going along and doing his ordinary day's work when the X-ray unit came round; or else, he felt a little short of breath or had a cough and went to have his chest radiologically examined; the result in either case was that he was found to have tuberculosis. The argument was put up that if he had been at rest all that time, he might not have developed tuberculosis, or else it might not have progressed as far as it had done. Dr. Hughes said that one might possibly agree with that, but it seemed a poor method of establishing a claim for compensation. Nowadays the courts did not usually allow such a claim; they generally required evidence of some special aggravating factor, such as fatigue from long hours or arduous work.

Dr. Joseph, in reply to Dr. McManis, said that asbestos bodies could be present in the lung without clinical and radiological evidence. Asbestos bodies were sometimes found in the sputum of symptomless asbestos workers; but it was hard to know whether the disease was there. However, as he had pointed out, even if the patient was removed from the dust, the condition tended to progress. Referring to spontaneous pneumothorax, Dr. Joseph pointed out that in the lung the alveoli mostly rested against each other, but in some situations they lay on a bronchovascular bundle. An unsupported alveolus might rupture, with resulting surgical emphysema and spontaneous pneumothorax. He had seen spontaneous pneumothorax with mediastinal emphysema occur in a woman in labour. One boy in his practice had had not less than eight spontaneous pneumothoraces; he worked in a produce store and some were associated with exertion, but the last occurred when he was lying on the floor watching television. Referring to cold as the cause of pneumonia, Dr. Joseph said that it had nothing to do with it. He instanced the Norwegians working on the Snowy Mountains Scheme, who liked to get under a very hot shower, and then run out and roll in the snow; they did not contract pneumonia.

Dr. Alexander, from the chair, thanked Dr. Hughes, an insurance medical officer, and Dr. Joseph, a practising physician, for their papers, on behalf of the Branch Council and those present.

Medical Matters in Parliament.

HOUSE OF REPRESENTATIVES.

THE following extracts from *Hansard* relate to the proceedings of the House of Representatives.

March 22, 1960.

Pharmaceutical Benefits.

MR. GALVIN: I direct a question to the Minister for Health. Will the honorable gentleman inform the House why the drug butazolidine is not included in the list of drugs available for the payment of 5s. on presentation of a doctor's prescription under the national health scheme? Does the fact that this drug has been restored to the list of drugs now available to pensioners under the pensioner medical service indicate the need for it to be included in the general scheme as this drug is one of those most regularly included in doctor's prescriptions?

DR. DONALD CAMERON: I understand the reason is that this drug possesses very considerable toxic qualities, and that the arrangements made for its supply are those laid down by the Pharmaceutical Benefits Advisory Committee who, in their judgement, consider that there are indications for its use for pensioners as a benefit, but not otherwise.

Hearing Aids for Pensioners.

MR. MCIVOR: I direct a question to the Minister for Social Services. Is it true that the only persons eligible for assistance in obtaining hearing aids are repatriation patients whose disability has been proved, war widows and children? If this is so, will the Minister extend the same assistance to age and invalid pensioners whose circumstances, the Minister will agree, do not allow them to obtain such an amenity.

MR. ROBERTSON: I appreciate the interest manifested by the honorable member for Gellibrand in the provision of hearing aids for those who require devices of the kind, but I suggest to him, with very great respect, that it is wrong to consider hearing aids in isolation. It would be necessary to consider artificial limbs, invalid chairs, spectacles, and in the case of those of us who need to hide our baldness from time, it would be necessary to consider other things. All these matters have been considered but, up to this point, the Commonwealth has decided that it would be much better in the interests of those who need assistance of the kind to leave it to the agencies that already provide it. How long that determination will continue I am not in a position to say, but I will consider what the honorable member for Gellibrand has suggested.

Hospital Benefits for Pensioners.

MR. CLYDE CAMERON: My question is directed to the Minister for Health. Is it true that in South Australia free public hospital treatment is given to pensioners without means, except those who contribute to a hospital benefits fund, and that in the case of these latter pensioners the hospitals adjust their accounts to make them correspond with the value of hospital benefits received by the pensioners, even in cases in which the benefits are more than £3 a day, which is the normal hospital charge? If these are facts, what benefit does a pensioner without means gain from paying into a hospital benefits fund? If there is no benefit to be gained by the pensioner, why did the Royal Adelaide Hospital, by instructions issued on 20th February and 28th February of this year, order its officers to make every effort to persuade pensioners to join a benefits organization?

DR. DONALD CAMERON: The question whether hospitals shall charge pensioners for treatment is, as I am sure the honorable gentleman appreciates, one to be settled by the State Governments. If the practices to which the honorable member refers are being followed in South Australia, I am afraid he must direct his enquiries to the Government of that State, not to me.

March 23, 1960.

Commonwealth Serum Laboratories.

MR. WARD asked the Minister for Health, upon notice:

1. What is the capital investment in the Commonwealth Serum Laboratories?

2. What is the present value of the laboratories and their assets?

3. What products produced at the laboratories are competitive on the Australian market with those produced by privately owned drug houses?

4. At what prices are these products marketed and are they fixed by agreement with the privately owned drug houses?

5. What is the retail price of each of the privately produced products.

6. What other drugs are produced in the Commonwealth Serum Laboratories?

7. Are these products retailed to the public and hospitals, etc., at a profit or on the basis of the cost of production?

8. What are the details in each case?

9. What amount has been expended upon research work in each year since the laboratories were established?

10. What has been the financial result of operations of the laboratories in each year since their establishment?

DR. DONALD CAMERON: The answers to the honorable member's questions are as follows:

1. The book value of the fixed assets as at 30th June, 1959, was £3,216,749.

2. The replacement value is not known.

3. Some classes of penicillin and insulin.

4. The prices of these products are determined having regard to cost of production and not by agreement with private interests.

5 and 6. The Commonwealth Serum Laboratories' price list containing some hundreds of items will be made available to the honorable member.

7 and 8. See 4 above.

9. and 10.

	Research	Financial Result of Operations.
	£	£
1956-57	55,485	136,908 (surplus)
1957-58	61,027	66,432 (surplus)
1958-59	94,809	212,007 (surplus)

Considerable research would be necessary to obtain this data back to the inception of the laboratories.

Antibody Milk.

MR. SWARTZ asked the Minister for Health, upon notice:

1. Is information available regarding experiments conducted in the United States of America with antibody milk produced to fight certain diseases?

2. Has some success been achieved in tests against some allergies and rheumatoid arthritis?

DR. DONALD CAMERON: The answer to the honorable member's questions is as follows:

1 and 2. It is understood that the idea behind the veterinary experiments is to isolate the organism causing rheumatoid arthritis, make a vaccine from it, and then introduce the vaccine into the cow's udder through the teat canal. The milk subsequently taken from the cow is then alleged to contain certain antibodies which will benefit the person suffering from the disease. Similarly, it is contended that pollens and other substances causing allergies can be handled in the same way and patients treated by drinking a minimum of a quart of "antibody milk" daily. The reasoning behind this research work is obscure and somewhat unscientific as, firstly, no germ has yet been identified as a cause of rheumatoid arthritis, and secondly the method of direct administration of a vaccine by injection is much more logical and certainly more likely to produce benefit than by the oral route, with the risk of inactivation in the stomach.

March 24, 1960.

Drugs.

MR. GALVIN: My question is directed to the Minister for Health. Has the honorable gentleman's attention been directed to a report that a drug sold in Australia under the trade name "Imferon" as an injection for the treatment of anæmia and rheumatoid arthritis is the same drug which the English branch of the British Medical Association has stated may cause cancer? If it is the same drug, what action, if any, is proposed concerning its sale in this country?

DR. DONALD CAMERON: This matter has come to my notice, and investigations are being made into it. I should point out to the honorable gentleman that there are very few drugs which have not some toxic property, and most drugs have to be used with caution.

Health and Medical Services.

MR. WARD asked the Minister for Health, upon notice:

What has been the expenditure on (a) free drugs, (b) medical services, (c) hospitalization, and (d) the administration in each year since the inauguration of the Commonwealth Health Scheme?

DR. DONALD CAMERON: The answer to the honorable member's question is as follows:

The amounts expended by the Commonwealth were—

Year Ended	(a) Pharmaceutical Benefits (Including Pensioner).	(b) Medical Benefits.	(b) Pensioner Medical Service.	(c) Hospital Benefits.	(d) Expenditure on Administration.
	£	£	£	£	£
June 30, 1946	—	—	—	1,111,292	13,830
June 30, 1947	—	—	—	4,380,296	10,614
June 30, 1948	—	—	—	4,448,015	25,772
June 30, 1949	149,037	—	—	5,885,446	40,282
June 30, 1950	304,689	—	—	6,320,164	44,568
June 30, 1951	2,930,163	—	75,511	6,535,628	140,471
June 30, 1952	7,685,046	—	1,036,225	6,683,106	195,109
June 30, 1953	7,215,309	—	1,739,953	7,223,241	323,733
June 30, 1954	9,229,413	1,434,166	2,115,539	8,330,053	300,341
June 30, 1955	10,739,467	4,209,495	2,516,077	9,320,603	333,513
June 30, 1956	11,887,434	5,413,320	2,874,364	9,552,944	383,426
June 30, 1957	11,716,825	6,146,029	2,998,886	9,813,283	393,111
June 30, 1958	15,033,989	7,085,524	3,198,791	10,823,096	386,574
June 30, 1959	20,972,797	7,779,451	3,806,457	14,802,290	431,833

These figures represent the expenditure directly relatable to the administration of medical and hospital benefits, the Pensioner Medical Service, and pharmaceutical benefits. A separate dissection is not kept of salaries and general expenses in respect of officers engaged partly on this work and partly on other duties.

Congresses.

SECOND ASIAN-PACIFIC CONGRESS OF CARDIOLOGY.

THE second Asian-Pacific Congress of Cardiology will be held in Melbourne from May 30 to June 3, 1960. The Organizing Secretary of the Congress is Dr. A. E. Doyle, Alfred Hospital, Melbourne, S.1; information relating to the congress may be obtained from him. Congress members are asked to register at the Congress Bureau at Menzies' Hotel. The full programme of the Congress is as follows.

TUESDAY, MAY 31; MORNING.

Theatre I: Epidemiology of Heart Disease.

9.30 a.m., "A Ten-Year Survey of Rheumatic Fever and Rheumatic Heart Disease in West Australia', C. Fortune; 9.50 a.m., "The Epidemiology and Prophylaxis of Rheumatic Heart Disease in India", S. Padmavati; 10.10 a.m., "Incidence of Various Ætiologies of Heart Disease in Bombay", K. K. Datey; 10.30 a.m., "Ætiological Survey of Cardiac Patients in Djakarta Visiting a Private Practice", Gan Tjong Bing; 11.10 a.m., "Bacterial Endocarditis in Sydney in the Last Ten Years', J. B. Hickie; 11.30 a.m., "Endomyocardial Fibrosis in Uganda", K. Somers; 11.50 a.m., "Some Problems of Differential Diagnosis in Cardiomyopathy", W. Brigden; 12.15 p.m., Panel discussion on epidemiology of heart disease; chairmen, M. L. Powell (Melbourne), S. Padmavati (New Delhi).

Theatre II: Cardiac Surgery—Technical and Experimental.

9.30 a.m., "The Sheep as an Experimental Animal in Surgical Science", J. Borrie; 9.50 a.m., "Experimental Hypothermia", T. T. Currie; 10.10 a.m., "Blood Coagulation Problems in Open-Heart Surgery", P. Fantl; 10.30 a.m., "Open-Heart Surgery Using Deep Hypothermia and Cardio-Pulmonary By-Pass", J. A. Simpson; 11.10 a.m., "Observations on Experimentally Produced Mitral Valve Incompetence', I. Monk; 11.30 a.m., "The Effect of Induced Cardiac Asystole on Ventricular Function", G. Stirling; 11.50 a.m., "Complete Heart Block as a Complication of Repair of Septal Defect (A Review of 62 Cases)", P. Ongley.

TUESDAY, MAY 31; AFTERNOON.

Theatre I: Rheumatic Heart Disease—Diagnosis and Treatment.

2 p.m., "Stenosis of Both Aortic and Mitral Valves: Clinical and Physiological Features", W. H. Abelmann (Boston, U.S.A.); 2.20 p.m., "Transmitral Diastolic Pressure Gradients in Normal Persons and Patients with Mitral Valve Disease", S. B. Roy (New Delhi); 2.40 p.m., "Surgical Treatment of Aortic Stenosis", D. Stuckey (Sydney); 3 p.m., "Surgery of Mitral Stenosis", C. S. Lazatin (Manila); 3.40 p.m., "Factors Leading to Errors in Diagnosis Following Cardiac Catheterization', H. F. Helmholz (Rochester, U.S.A.); 4 p.m., "Cardiac Rehabilitation: A Challenge", E. G. Galea (Brisbane); 4.30 p.m., Panel discussion on rheumatic heart disease; chairmen, J. B. Lowe (Auckland), S. B. Roy (New Delhi).

Theatre II: Physiology of Vessels.

2 p.m., "Coronary Insufficiency and Coronary Branch Insufficiency", E. Kimura (Tokyo); 2.20 p.m., "Effect of Dopamine (3-Hydroxytyramine) Upon the Systemic Pulmonary and Coronary Hæmodynamics and Metabolism of Intact Dog", G. M. Maxwell (Adelaide); 2.40 p.m., "Experimental Observations on Effects of Sympathetic Nerve Stimulation and Sympathomimetic Drugs on Coronary Circulation and Electrocardiogram", S. Hayase (Kyoto); 3 p.m., "The Effect of Serotonin on Blood Pressure in Pulmonary and Systemic Circulation", M. Toor (Israel); 3.40 p.m., "Modification of Secondary Lesions in Experimental Hypertension by Control of Blood Pressure", J. V. Hodge (Dunedin); 4 p.m., "Neural Control of the Cardio-Vascular System", R. F. Rushmer (Seattle, U.S.A.).

WEDNESDAY, JUNE 1; MORNING.

Theatre I: Coronary and Arterial Disease.

9.30 a.m., "Ischæmic Heart Disease", C. Burns (Wellington); 9.50 a.m., "The Possible Role of Obesity in Ischæmic Heart Disease", H. M. Whyte (Sydney); 10.10 a.m., "Subendocardial Infarction", E. H. Roche, (Auckland); 10.30 a.m., "Genetic Factors in the Pathogenesis of Coronary Disease", J. Stokes (Boston); 11.10 a.m., "A Clinical Study on the Ischæmic States of the Extremities", K. Ishikawa (Tokyo); 11.30 a.m., "Arterial Grafting for Occlusive Arterial Disease of the Lower Limbs", A. J. Barnett (Melbourne); 11.50 a.m., "Intrathoracic Aortic Aneurysms", H. M. J. Windsor (Sydney); 12.15 p.m., Panel discussion on coronary and arterial disease; chairmen, J. Halliday (Sydney), A. M. Samia (Manila).

Theatre II: Pulmonary Circulation.

9.30 a.m., "Interrelationships Between Blood Flow, Pressure and Volume in the Pulmonary Vascular Bed in Man", A. D. Jose (Sydney); 9.50 a.m., "Ventilatory Response to Exercise in Heart Disease", J. G. Sloman (Melbourne); 10.10 a.m., "Bronchial Obstruction: Its Significance in Pulmonary Hypertension", H. J. H. Colebath (Sydney); 10.30 a.m., "Pulmonary Hypertension in Patients with Pure or Predominant Mitral Incompetence", W. A. Seldon (Sydney); 11.10 a.m., "The Importance of Vascular Distensibility in Circulatory Dynamics", W. R. Milnor (Baltimore); 11.30 a.m., "A Note on the Murmur of Tricuspid Incompetence", O. B. Tofler (Perth); 11.50 a.m., "Hæmodynamics Effects of Squatting", T. V. O'Donnell (San Francisco).

WEDNESDAY, JUNE 1, AFTERNOON.

Theatre I: Instrumental Methods.

2 p.m., "Electrocardiographic Changes During a Two-Hour Hike in a Hot Climate Under Different Conditions of Hydration", M. Toor (Petah Tiqva); 2.20 p.m., "Intra-arterial Block", Du Bong Lee (Seoul); 2.40 p.m., "Trans-Septal Left Heart Catheterization', E. Braunwald (Bethesda, U.S.A.); 3 p.m., "Factors Involving the Shaping of Arterial Pressure Pulses", M. G. Taylor (Sydney); 3.40 p.m., "The Estimation of Valvular Incompetence in the Mammalian Circulation", P. I. Korner (Sydney); 4 p.m., "Successive Measurement of Cardiac Output by External Monitoring", H. Ueda (Tokyo); 4.20 p.m., "The Diagnosis of Left Ventricular Hypertrophy", N. Kimura (Kurume).

Theatre II: Lipids and Thrombosis.

2 p.m., "Cholesterol Levels and Coronary Disease in New Zealand Males", J. D. Hunter (Dunedin); 2.20 p.m., "Serum Lipids, Blood Clotting and Fibrinolysis in Natives of New Guinea and Australia", E. Goldrick (Sydney); 2.40 p.m., "Studies in Atherosclerosis", M. Murakami (Kanazawa); 3 p.m., "The Inhibitory Effect of Platelets on Heparin-Induced Clearing', P. J. Nestel (Melbourne); 3.40 p.m., "Non-Fatty 'Arteriosclerogenic Substances' and Success in Producing Atherosclerosis by These Substances", T. Shimamoto (Tokyo); 4 p.m., "Lipid Tolerance in Coronary Disease", G. V. Hall (Sydney); 4.20 p.m., "Lipoprotein Lipase Activity of Human Blood Plasmas", R. J. Havel (San Francisco).

THURSDAY, JUNE 2, MORNING.

Theatre I: High Blood Pressure.

9.30 a.m., "The Pathogenesis of Inherited Spontaneous Hypertension in Rats", F. H. Smirk (Dunedin); 9.50 a.m., "A Clinical Survey of Incidence of Hypertension in Rural Populations of Kanpur and in Industrial Population of Kanpur (U.P.)", K. N. Gour (Kanpur); 10.10 a.m., "The Distribution of Blood Pressure of Koreans", K. H. Cho (Seoul); 10.30 a.m., "Peripheral Vascular Action of Reserpine in Man", R. F. Whelan (Adelaide); 10.50 a.m., "Bretylium Tosylate in the Treatment of Hypertension" G. E. Bauer; 11.35 a.m., "Sites of Action of Hypotensive Drugs", J. W. McCubbin (Cleveland); 11.55 a.m., Panel discussion on therapeutics; chairmen, Sir Horace Smirk (Dunedin), M. Toor (Israel).

Theatre II: Myocardial Metabolism I.

9.30 a.m., "The Significance of Plasma Transaminase Activity in Ischæmic Heart Disease", E. N. O'Brien (Melbourne); 9.50 a.m., "The Sodium and Potassium Content of Normal Human Hearts", C. Dayrit (Manila); 10.10 a.m., "The Occurrence of Cardiac Stimulant Activity in Extracts of Mammalian Tissues", R. H. Thorp (Sydney); 10.30 a.m., "The Cardiotonic Principle in Spleen", L. B. Cobbin (Sydney); 10.50 a.m., "Some Supporting Evidence to the 'Chain Doublet' Hypothesis (Maekawa) Concerning Heart Action Potential", M. Maekawa (Kyoto).

THURSDAY, JUNE 2; AFTERNOON.

Theatre I: Myocardial Metabolism II.

2 p.m., "Myocardial Metabolism in Hypertension, Coronary Sclerosis and Diabetes Mellitus", T. Kobayashi (Tokyo); 2.20 p.m., "Cell Membrane Permeability as a Basis of Glycoside Activity", W. Nayler (Melbourne); 2.40 p.m., "The Excretion and Metabolism of Cardiac Glycosides with Observations on Possible Clinical Significance", S. E. Wright (Sydney); 3 p.m., "Trace Metals and Myocardial Enzymes", G. Griffith (Los Angeles); 3.40 p.m., "Differential Effects of Metabolic Inhibitors on Sinus, Atrial Ventricular and Conus Action Potentials", W. V. Macfarlane (Canberra); 4 p.m., "The Pharmacology of Cardiac Innervation", M. J. Rand (Sydney).

Theatre II: Congenital Heart Disease.

2 p.m., "Teratoma of the Pericardium", K. Soekarjo (Djakarta); 2.20 p.m., "The Diagnosis of Atrio-Ventricular Canal Defects", J. B. Lowe (Auckland); 2.40 p.m., "Clinical and Morphological Variations in the Tetralogy of Fallot", K. I. Vytilingam (Vellore); 3 p.m., "Primary Pulmonary Hypertension in Infancy and Childhood", J. F. Farrar

(Sydney); 3.40, "Agenesis of a Main Branch of the Pulmonary Artery", A. Murphy (Brisbane); 4 p.m., "Rubella in Pregnancy and Congenital Heart Disease", I. S. Wallmann (Perth); 4.20 p.m., "Prolonged Veno-Arterial Pumping for the Relief of Cardiac Stress", S. B. Roy (New Delhi).

FRIDAY, JUNE 3; MORNING.

Theatre I: Cardiac Surgery.

9.30 a.m., "The Results of Repair of Atrial Septal Defect Using the Atrial Well Method", B. G. Barratt-Boyes (Auckland); 9.50 a.m., "The Surgery of Atrial Septum Primum Defects", R. Nicks (Sydney); 10.10 a.m., "The Problems of Aortic Stenosis", K. N. Morris (Melbourne); 10.30 a.m., "Pericardial Autografts in Heart with Description of By-Pass Technique for Replacement of Mitral Valve Through Left Chest", L. R. Sauvage (Seattle, U.S.A.); 11.10 a.m., "Corrected Transposition of Great Vessels: A Study of 33 Cases", P. Ongley (Rochester, Minn.); 11.30 a.m., "Patent Ductus Arteriosus in Infancy", A. W. Venables and N. A. A. Myers; 11.50 a.m., "Surgical Management of Coarctation of the Aorta", C. J. Officer Brown (Melbourne); 12.15 p.m., Panel discussion on congenital heart disease; chairmen, C. J. Officer Brown (Melbourne), K. I. Vytilingam (Vellore).

Theatre II: Coronary Vascular Disease.

9.30 a.m., "The Interpretation of Isotopic Lipid Tolerance Curves as Studied in Coronary Disease", E. P. George (Sydney); 9.50 a.m., "Arteriosclerosis and Gall-Stones in the Philippines", A. G. Sison (Manila); 10.10 a.m., "The Diagnosis of Ventricular Paroxysmal Tachycardia", C. J. McRae (Melbourne); 10.30 a.m., "Coronary Angiography", J. G. Sloman (Melbourne); 11.10 a.m., "Coronary Artery Disease in Geelong", J. M. Agar (Geelong); 11.30 a.m., "Anastomosis in the Coronary Circulation", W. Laurie (Perth); 11.50 a.m., "Establishing a Collateral Coronary Circulation by Hypoxia", R. Heilig (Jaipur).

FRIDAY, JUNE 3; AFTERNOON.

Theatre I: Heart Failure.

2 p.m., "Œdema in Cardiac Failure", T. E. Lowe (Melbourne); 2.20 p.m., "The Renal Clearance and the Venous Pressure in Congestive Heart Failure", M. Maekawa (Kyoto); 2.40 p.m., "Mechanism, Prognosis and Treatment of Shock Following Myocardial Infarction", R. W. Gunton (Toronto); 3 p.m., "Regulation of Electrolytes in the Management of Cardiac Failure", E. V. Newman (Nashville, U.S.A.); 3.20 p.m., "The Exchange of Labelled Serum Protein Between Serum and Œdema in Congestive Heart Failure", R. S. Ross (Baltimore); 4 p.m., "Digitalis", J. McMichael (London); Closing remarks, J. K. Maddox (Sydney).

Out of the Past.

SALARY FOR ASSISTANT.[1]

[To the Editor of the *Australasian Medical Gazette*, December 20, 1900.]

SIR. Can you inform me who is the member of the profession who dares to offer the princely sum of £150 per annum for an assistant? I am glad he seems to have some difficulty in securing one, judging from the frequency of his advertisement which appears in the Sydney Morning Herald. I think that the name of the so called liberal medical gentleman should be printed and placed on a black list so that we may know who he is and give him the treatment which he so richly deserves. I think that a man who tries to sweat his fellow practitioner is certainly guilty of an offence, and should be tabooed by the other members of the profession. What is the use of forming Investigation Committees to enquire into the sweating of the profession by the public when we have men amongst ourselves trying to sweat their fellows? I say, let us look at home first. (The advertisement referred to appears at foot).

Yours etc.,
MEDICUS.

Medical—Wanted, Legally-qualified Assistant for N.S. Wales railway town £150 all found.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

GENERAL PHARMACEUTICAL BENEFITS.

SIR: We feel that a voice should be raised against the seemingly unwarranted exclusion of pempidine tartrate ("Perolysen") from the pharmaceutical benefits. To those treating hypertension, a wide range of hypotensive drugs is necessary to achieve satisfactory results because of the individual variation in response. There are several other points which favour the inclusion of pempidine in the benefits scheme.

The two potent ganglion-blocking preparations in current use which are satisfactory when taken orally because of complete absorption are mecamylamine ("Mevasine") and pempidine. Both have been shown to produce a smooth and predictable hypotensive effect (Doyle, Murphy and Neilson[1]; Harrington, Kincaid-Smith and Milne[2]) which was not achieved by earlier oral preparations. Mecamylamine and pempidine have also been shown to be effective and life-saving in malignant hypertension (Doyle *et alii*;[1] Harrington *et alii*;[2] Harrington, Kincaid-Smith and McMichael[3]). Pempidine has certain advantages over mecamylamine (Harrington *et alii*[2]); because of its more rapid excretion, serious complications such as paralytic ileus are less likely to occur and, even more important, are of short duration. The neurological complications of psychosis and tremor which have been reported with mecamylamine (Schneckloth, Corcoran, Dunstan and Page;[4] Harrington and Kincaid-Smith[5]) have not been observed with pempidine. Both these complications may prove fatal. In spite of the development of new drugs which do not produce the side-effects of parasympathetic blockade, pempidine and mecamylamine are still considered the drugs of choice for the treatment of malignant hypertension and hypertension with coexisting myocardial and cerebral ischæmia.

For all these reasons, we think it highly advisable that pempidine should be made available as an unrestricted pharmaceutical benefit as soon as possible.

Yours, etc.,
K. F. FAIRLEY,
P. S. KINCAID-SMITH.

421 St. Kilda Road,
Melbourne, S.C.2.
May 2, 1960.

SIR: Attention should be drawn to the fact that at least one prescription of the Pharmaceutical Benefits is a potentially dangerous remedy. I refer to Compound Calamine Liniment on page 6, Section 3, of the Prescribers' List. This formula contains phenol (B.P.) and camphor (B.P.), a combination which, in the presence of water, forms a caustic solution liable to superimpose a chemical dermatitis on the condition for which it has been prescribed.

As a dermatologist, the writer has been aware of this for some time, and one or both of these ingredients should be omitted.

Yours, etc.,
IVAN M. WARTZKI.

81 Collins Street,
Melbourne.
May 3, 1960.

SIR: I am heartily fed up with the following: (i) Writing prescriptions in duplicate. (ii) Constantly referring to the "good book of anomalies" to make sure I am not contravening any of its many petty regulations. (iii) Deciding whether my patients fall into the category of a "specified purpose". (iv) Deciding whether it is cheaper for the patient to buy the drugs outright, or get them "free". (v) Having to fill in all the details of the patient's age, address, etc., when such is unnecessary. (vi) Many other time-consuming irritations associated with the present Pharmaceutical Benefits Scheme.

Also, I wonder whether the Government is saving money at the expense of Australia and its public, and I feel

[1] *Brit. med. J.*, 1956, 2:1209.
[2] *Lancet*, 1958, 2:6.
[3] *Brit. med. J.*, 1959, 2:969.
[4] *J. Amer. med. Ass.*, 1956, 162:868.
[5] *Lancet*, 1958, 1:499.

sure that this set of regulations and directives, with its associated penalties and punishments if one should transgress, is but the "thin end of the wedge" for a completely socialized medical scheme which will only bring with it more frustration and hardship than we have ever dreamed of, and a consequent lowering of our standards to that of "clinical clerks" under direct bureaucratic control.

Does anyone else feel like this? If so, please write in duplicate, stating name, age, address, and endorsing each copy, if applicable, with the words "For the specified purpose of preserving our medical identity and individuality".

Yours, etc.,

202 Blaxland Road, Ryde, N.S.W.
April 26, 1960.

JOHN B. GRAY.

TUMOUR CELLS IN THE BLOOD.

SIR: Your editorial on the above subject (MED. J. AUST., April 30, 1960) is a most informative communication on the present state of knowledge of this subject. It omitted to mention the fact that a resident medical officer of the Melbourne Hospital (T. R. Ashworth) first observed the phenomenon (1869). Ashworth's original drawings and article may still be read in the *Australian Medical Journal* for that year, which is in the British Medical Association's library in Sydney.

Ashworth obviously had considerable difficulty in obtaining pathological assistance with regard to the nature of the cytæmic tumour. We find ourselves in similar difficulties because, at the Special Unit of the New South Wales State Cancer Council, we have a number of projects planned which depend for their execution on a ready method of identification of cancer cells in the blood. Many approaches have been made to hæmatologists and pathologists in Sydney, who assure us that the identification of these cells is not only tedious, difficult and uncertain, but that interpretation also may be the subject of dispute.

We have therefore maintained a vigorous correspondence with overseas centres in the hope of finding what your editorial describes as "a fairly ready method of identification". If we are unduly pessimistic in this matter, I would very much appreciate it if any of your readers could bring to our notice a reliable and practical method of estimating circulating tumour cells in the blood, comparable with the ordinary procedures of hæmatology.

For any of your readers who are interested in our views on this subject, publication No. 3 (1960) of the New South Wales State Cancer Council is readily available and may prove of interest.

Yours, etc.,

149 Macquarie Street, Sydney.
May 4, 1960.

K. W. STARR.

Post-Graduate Work.

AUSTRALIAN ASSOCIATION OF NEUROLOGISTS: SCIENTIFIC MEETING.

THE Australian Association of Neurologists will hold a scientific meeting at the Wood Jones Lecture Theatre, Anatomy School, University of Melbourne, on Tuesday, May 24, 1960. Each paper will occupy 20 minutes, and will be followed by a discussion. The programme is as follows.

10 a.m., "Anticonvulsants in the Treatment of Facial Pain", Dr. R. H. S. Rischbieth; 10.30 a.m., "The Neurophysiology of Kuru", Dr. L. R. Rail; 11.30 a.m., "On Cerebellar Localization Using Positive Contrast", Dr. A. Fisher; 12 noon, "Olivo-Ponto-Cerebellar Degeneration with Extrapyramidal Involvement", Dr. B. Turner; 12.30 p.m., "Familial Myoclonic Epilepsy and its Association with Cerebellar Disorder" (including 16 mm. cinematographic film), Dr. J. W. Lance and Dr. K. B. Noad; 2.30 p.m., "Some Observations on Parkinsonism. (Thoughts and Questions about the Pathological Anatomy and Physiology of Certain Aspects of the Various Forms of Parkinsonism)", Dr. G. Selby; 3 p.m., "The Symptomatology of Multiple Sclerosis Based on the Review of 535 Patients", Dr. J. M. Sutherland; 4 p.m., "Experimental Encephalomyelitis", Dr. R. Anderson and Dr. G. Szego; 4.30 p.m., "Recent Experiences in the Investigation and Treatment of Temporal Lobe Epilepsy, Particularly in Relation to Surgery", Dr. Eric Davis.

The Honorary Secretary is Dr. J. A. Game, 61 Collins Street, Melbourne.

AUSTRALIAN RHEUMATISM COUNCIL.

Fellowship in Rheumatic Diseases.

APPLICATIONS are invited from graduates in Medicine with not less than four years' post-graduate experience for a Fellowship in Rheumatic Diseases tenable in the United Kingdom commencing in 1960. This Fellowship has been made available by the Empire Rheumatism Council to enable a suitably qualified graduate to obtain further research and/or clinical training in the field of the rheumatic and allied diseases. Preference will be given to an applicant who holds a higher degree or diploma. Applicants should have worked in a clinical department and have assisted in the research work carried out in such a department.

Consideration will be given to two types of applicant: younger graduates of the registrar grade who may wish to carry out research or clinical training leading to senior appointments in medicine, preferably rheumatology, or alternatively more senior graduates interested in the rheumatic diseases. The former would be required to spend a minimum of nine months in a suitable institution or clinic. The senior type of Fellow might hold the Fellowship for a shorter period.

The remuneration will be at the rate of £1000 stg. per annum. Applications should be addressed to the Honorary Secretary, Australian Rheumatism Council, c.o. M.L.C. Ass. Ltd., Victoria Cross, North Sydney. Applicants should give full particulars of academic qualifications, post-graduate experience, past and present appointments, and publications, and should nominate two referees. Applications will close on June 3, 1960. Further particulars may be obtained from Dr. Selwyn Nelson, Medical Director, Australian Rheumatism Council, at the above address.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Conference at Katoomba.

THE Post-Graduate Committee in Medicine in the University of Sydney, in conjunction with the Blue Mountains Medical Association, will hold a post-graduate week-end conference in the ballroom at the Carrington Hotel, Katoomba, on Saturday and Sunday, June 25 and 26, 1960. The programme is as follows:

Saturday, June 25: 2 p.m., registration; 2.30 p.m., "Diagnosis of Lesions of the Large Bowel and Cases of Surgical Interest with Slides", Dr. Alexander Johnson; 4.30 p.m., "Cardiac Emergencies", Dr. J. G. Richards.

Sunday, June 26: 10.15 a.m., "The Acute Abdomen with Special Reference to Visceral Trauma and Intestinal Obstruction", Dr. Alexander Johnson; 11.45 a.m., "The Psychosomatic Approach to Disease", Dr. David Ross; 2.15 p.m., "Acute Chest Emergencies and their Management", Dr. J. G. Richards; 3.45 p.m., "Asthma", Dr. David Ross.

The fee for lectures is four guineas, which includes a post-graduate fee of three guineas, and those wishing to attend the course are requested to communicate as soon as possible with Dr. A. W. Raymond, Honorary Secretary, the Blue Mountains Medical Association, "Sunningdale", Great Western Highway, Wentworth Falls. Telephone: Wentworth Falls 81.

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

PROGRAMME FOR JUNE, 1960.

THE Melbourne Medical Post-Graduate Committee announces the following programme for June, 1960.

Overseas Visitors.

Dr. Wallace Brigden, M.A., M.D., F.R.C.P., cardiologist and physician of the London Hospital and the National Heart Hospital, will attend the Asian-Pacific Congress of Cardiology from May 30 to June 4. After this he will

engage on the following programme. On Monday, June 6, he will lecture on "Syncope" at 12 noon at the Royal Melbourne Hospital, and at 8.15 p.m. in the Medical Society Hall he will lecture on "Current Problems in the Medical Treatment of Heart Disease". On Wednesday, June 8, he will speak on "Non-Coronary Myocardial Disease" at 1.15 p.m. at the Alfred Hospital; this lecture is for the Resident Medical Officers' Clinical Society.

Dr. Brigden will undertake the following hospital visits: Monday, June 6, Royal Melbourne Hospital; Tuesday, June 7, St. Vincent's Hospital in the morning, Prince Henry's Hospital in the afternoon; Wednesday, June 8, the Alfred Hospital.

Professor Bryan McFarland, M.D., F.R.C.S., Professor of Orthopædic Surgery, Liverpool, will visit Melbourne from June 6 to 17, as the guest of the Australian Post-Graduate Federation in Melbourne. The programme is as follows:

The lectures set out below will be given at 8.15 p.m. in the Medical Society Hall: Tuesday, June 7, "Limp in Childhood"; Thursday, June 9, "Osteoarthritis of the Hip. I"; Tuesday, June 14, "Osteoarthritis of the Hip. II"; Thursday, June 16, "Internal Derangement of the Knee".

The following hospital visits will all be made in the morning, with the exception of those on June 15 and 17, which will be made in the afternoon: Monday, June 6, St. Vincent's Hospital; Tuesday, June 7, and Wednesday, June 8, Royal Children's Hospital; Thursday, June 9, and Friday, June 10, the Alfred Hospital; Tuesday, June 14, Royal Melbourne Hospital; Wednesday, June 15, Repatriation General Hospital, Heidelberg; Thursday, June 16, Royal Melbourne Hospital; Friday, June 17, Prince Henry's Hospital.

The fees for attendance as the evening lectures listed above are at the rate of 15s. per lecture, but annual subscribers to the Committee may attend without further charge.

Course in Medicine.

The honorary medical staff of the Royal Melbourne Hospital will conduct a post-graduate course in medicine suitable for candidates sitting for the degree of M.D. (Melbourne) or the M.R.A.C.P. diploma, from June 6. This course will run for eight weeks; sessions will be held from 12 noon to 5.15 p.m. on Mondays to Fridays, and from 9.30 a.m. to 12 noon on Saturdays. The course will consist of lectures, discussions and case presentations, seminars on radiology, morbid anatomy, clinical pathology, electrocardiography, modern investigatory techniques, etc.

Enrolments, together with the fee of thirty guineas, should be sent to the Committee at the address given below, as soon as possible.

Course in Pathology for Primary F.R.A.C.S. Examination.

Enrolments are due by June 20 for the course in pathology for the Primary F.R.A.C.S. examination, commencing on July 4. This course will be conducted at the University of Melbourne on Mondays and Wednesdays at 1.30 p.m. for six weeks. The fee is seven guineas.

Country Courses.

Colac.—At Colac, on June 25, the following course will be given: 3.45 p.m., "Dysmenorrhœa", Dr. Ian McDonald; 5.15 p.m., "Diagnosis and Management of Depressive States", Dr. John Cade. Dr. R. R. Sobey, 6 Spence Street, Warrnambool, is the local secretary. Attendance is by a fee of 15s. per lecture, or by annual subscription to the Committee.

Albury.—Attention is drawn to the course at Albury on June 18 and 19 which will be conducted by the Post-Graduate Committee in Medicine in the University of Sydney, in conjunction with the Melbourne Medical Post-Graduate Committee and the Border Medical Association.

Recorded Lectures.

The following recorded lectures have been added to the Committee's library on ten-inch microgroove disks, with accompanying illustrations, and may be borrowed without charge on request: "The Nature of the Staphylococcus Problem", by Dr. Hattie Alexander, of New York; March, 1960; two disks, five slides (2 in. by 2 in.); "Physiological Principles in the Management of Heart Failure", by Professor John McMichael, of London; March, 1960; two disks, 19 slides (2 in. by 2 in.).

Address.

The address of the Melbourne Medical Post-Graduate Committee is 394 Albert Street, East Melbourne. Telephone: FB 2547.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED APRIL 16, 1960.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	1	1	..	1(1)	..	..	..	3
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	6	..	6
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	12(2)	7(6)	2(2)	..	1	..	2	..	24
Diphtheria	..	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	..	..	..	..	3(3)	..	3	..	6
Encephalitis	..	..	..	1(1)	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	1	..	..	1
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	39(24)	15(9)	8(1)	..	5(3)	2	..	1	70
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	6	..	1	..	..	..	..	..	7
Malaria	..	..	2(2)	..	..	..	..	..	2
Meningococcal Infection	..	1(1)	..	..	1(1)	..	..	..	2
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	2(2)	..	..	..	2
Paratyphoid	..	..	..	..	1	..	..	..	1
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	1(1)	..	..	..	..	..	..	..	1
Puerperal Fever	..	1	..	..	..	..	..	..	1
Rubella	..	8(7)	..	..	1(1)	..	..	..	9
Salmonella Infection	..	..	..	..	1(1)	..	..	..	1
Scarlet Fever	9(5)	13(6)	2(2)	3(2)	2(2)	..	..	..	29
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	3(3)	..	..	..	..	..	3
Trachoma	..	..	..	..	2	..	..	..	2
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	22(10)	13(11)	12(7)	5(4)	8(3)	3	2	..	65
Typhoid Fever	..	1	..	..	..	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Australian College of General Practitioners.

NEW SOUTH WALES FACULTY.

Clinical Week-End.

THE first clinical week-end for 1960 of the New South Wales Faculty of the Australian College of General Practitioners will commence on Friday, June 3, at the Hotel Florida, Terrigal, with a late afternoon party at 5.30 p.m., and will end after luncheon on Sunday, June 5. During the week-end Sir William Morrow will address the College on "Modern Trends in Therapeutics", Mr. Frank Mills will discuss "The Management of Post-Operative Complications of Abdominal Surgery", and Dr. Harold Hodge will speak on "Safe Anæsthesia". As usual, there will be one of the formal lectures on the Sunday morning, as well as a session "Ask the Experts", featuring Sir William Morrow, Mr. Frank Mills and Dr. Harold Hodge.

College Forum Session.

This is a comparatively new session at week-ends. One hour is reserved for members of the College to express their views on any matter concerning College affairs or medical practice.

Notes and News.

Nuffield Travelling Fellowship for General Practitioners of Australia.

As part of its programme for the advancement of health, the Nuffield Foundation, in cooperation with the College of General Practitioners, is prepared to award to a citizen of Australia one Fellowship to enable a general medical practitioner of outstanding ability, preferably between the ages of 35 and 45 years, to undertake approved study overseas for a period of six consecutive months during 1961 in some subject of importance to general practice. The value of the Fellowship award will cover for the Fellow (and, if required, also for his wife) tourist travel by air, and other fares at tourist rates, together with an adequate subsistence allowance. The closing date for applications is September 30, 1960. They should be sent to the Honorary Secretary, Australian College of General Practitioners, 203 Macquarie Street, Sydney, New South Wales, from whom details of conditions and application forms can be obtained.

Medical Practice.

NATIONAL HEALTH ACT.

THE following notice is published in the *Commonwealth of Australia Gazette*, No. 29, of April 28, 1960.

NATIONAL HEALTH ACT 1953-1959.

Notice in Pursuance of Section 134a.

Notice is hereby given that following investigation and report by the Medical Services Committee of Inquiry for the State of New South Wales, concerning the conduct of Ronald James Jennaway of 28 Mary Street, Auburn, medical practitioner, I, Donald Alistair Cameron, Minister of State for Health, did on the seventh day of April, 1960, reprimand the said Ronald James Jennaway for conduct which is a breach of the National Health (Pharmaceutical Benefits) Regulations.

Dated this seventh day of April, 1960.

DONALD A. CAMERON,
Minister of State for Health.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Carew, Bernard John, M.B., B.S., 1956 (Univ. Sydney), 91 Jonson Street, Byron Bay, N.S.W.

Deaths.

THE following death has been announced:

ROCHE.—Cedric Watson Gray Roche, on May 5, 1960, at Magnetic Island.

Diary for the Month.

MAY 24.—New South Wales Branch, B.M.A.: Hospitals Committee.
MAY 24.—Tasmanian Branch, B.M.A.: Southern Subdivision.
MAY 25.—Western Australian Branch, B.M.A.: General Meeting.
MAY 25.—Victorian Branch, B.M.A.: Branch Council.
MAY 26.—New South Wales Branch, B.M.A.: Branch Meeting.
MAY 26.—Tasmanian Branch, B.M.A.: Northern Subdivision.
MAY 26.—South Australian Branch, B.M.A.: Scientific Meeting (Listerian Oration).
MAY 27.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

Authors of papers are asked to state for inclusion in the title their principal qualifications as well as their relevant appointment and/or the unit, hospital or department from which the paper comes.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this Journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in Australia can become subscribers to the Journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £6 *per annum* within Australia and the British Commonwealth of Nations, and £7 10s. *per annum* within America and foreign countries, payable in advance.